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## Table of Contents

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ORIGINAL ARTICLES—	PAGE.	SPECIAL ABSTRACT—	PAGE.
Listerian Oration—"A Study of Certain Principles of Mechanics in the Production and Treatment of Fractures," by FAY MACLURE, F.R.C.S.	715	Neonatal Mortality .. . . .	736
"The Skin Tolerance for Pressure and Pressure Sores," by HUGH C. TRUMBLE, M.C., M.B., B.S., F.R.C.S., F.C.S.A. . . . .	724	<b>SPECIAL ARTICLES ON DIAGNOSIS—</b>	
"Some Aspects of Bronchial Asthma and Allied Conditions," by G. C. WILLCOCKS, M.B., M.R.C.P. . . . .	726	Glaucoma .. . . .	739
<b>REPORTS OF CASES—</b>		<b>BRITISH MEDICAL ASSOCIATION NEWS—</b>	
"A Dangerous Typhoid Carrier," by W. J. PENFOLD, M.B., Ch.M., D.P.H., B.Hyg., M.R.C.S., L.R.C.P., and A. HYAMS .. . . .	728	Medico-Political .. . . .	741
<b>REVIEWS—</b>		<b>OBITUARY—</b>	
Progress in Pædiatrics .. . . .	730	Neville Bamanji Gandeia .. . . .	742
Australian Literature .. . . .	730	George Percival Stanley .. . . .	742
The Slit Lamp .. . . .	730	<b>CORRESPONDENCE—</b>	
Massage and Exercises .. . . .	730	Talipes Equinovarus Congenitalis .. . . .	742
<b>LEADING ARTICLES—</b>		Clinical Pathologists .. . . .	743
Medical History .. . . .	731	The Function of the Gall Bladder .. . . .	744
<b>CURRENT COMMENT—</b>		<b>BOOKS RECEIVED .. . . .</b>	744
Pneumonia and Pneumococci .. . . .	732	<b>DIARY FOR THE MONTH .. . . .</b>	744
Quinsy and Diphtheria .. . . .	733	<b>MEDICAL APPOINTMENTS .. . . .</b>	744
<b>ABSTRACTS FROM CURRENT MEDICAL LITERATURE—</b>		<b>MEDICAL APPOINTMENTS VACANT, ETC. .. . . .</b>	744
Surgery .. . . .	734	<b>MEDICAL APPOINTMENTS: IMPORTANT NOTICE</b>	744
		<b>EDITORIAL NOTICES .. . . .</b>	744

### Listerian Oration.<sup>1</sup>

#### A STUDY OF CERTAIN PRINCIPLES OF MECHANICS IN THE PRODUCTION AND TREATMENT OF FRACTURES.<sup>1</sup>

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PHYSIOLOGICAL and pathological processes enter largely into a consideration of the problems associated with fracture, but as the lesion is produced in the first instance by purely physical force, it will readily be recognized that a knowledge of the mechanical factors involved is of the greatest importance for a proper understanding of the subject.

By a careful study of the circumstances leading to a fracture it is possible in many cases to foretell what the X ray examination will reveal and, conversely, an examination of the skiagram will frequently allow of the reconstruction of the accident.

It may be taken as an axiom that the force used in the reduction of a displaced fracture must be exerted in a direction exactly opposite to that which produced the fracture. For example, the green stick fracture which has been caused by bending, must be corrected by bending in the opposite direction; the long oblique fracture which has been caused by a shearing strain has to be treated by extension; the depressed fracture by elevation; the abduction fracture by adduction; the torsion fracture by untwisting, and so on.

An investigation into the mechanical principles governing the production of fractures and their

<sup>1</sup> Delivered before the South Australian Branch of the British Medical Association on May 29, 1930.

subsequent repair will be of interest and value in treatment. And such an inquiry forms the basis of this study.

#### Transverse and Oblique Fractures.

The first example of what is intended lies in a comparison of the transverse and long oblique fractures as regards the mechanical phenomena presented by them and of the effects of these during treatment. The first stages in the repair of a fracture include the healing and subsequent contraction of the traumatized soft parts surrounding the broken bone. This is the process which gradually limits the mobility of the fragments and leads to early stiffening of the fracture. It is in the nature of a circular binding or whipping such as is used in splicing a broken fishing rod or golf club shaft (see Figure 1).

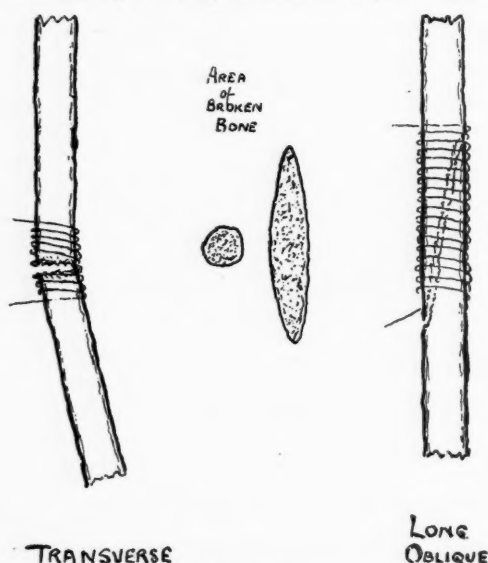


FIGURE 1.

It is obvious from Figure 1 that when the crack is oblique and therefore extensive, the binding or whipping will be more effective than when the fracture is transverse; and consequently the pre-natural mobility more quickly disappears; and also as the splicing is over a greater length, the resistance to bending will be greater. Moreover, the area of traumatized bone from which ossification proceeds, is at its minimum in the transverse fracture, whereas in the long oblique it approaches its maximum. So the prognosis as regards primary stiffening, freedom from angular deformity and early and strong bony union is very much better in the case of the long oblique than in that of the transverse fracture. So whilst the management of a transverse fracture of the neck of the femur will fully test any surgeon's skill and is always a slow and tedious process, frequently resulting in non-union, the long intertrochanteric fracture requires little treatment and always unites firmly in a comparatively short time.

The mechanical principle that lies behind this illustration should be borne in mind in the operative treatment of ununited fractures of long bones. The chance of success will be greater if opportunity be taken to convert a short transverse into a long oblique junction. This procedure necessarily involves the sacrifice of some length of limb. In the upper limb, and especially in the case of the humerus, where length is of secondary importance, the bone ends should always be trimmed to form a long junction, whilst in the lower limb the disadvantage of shortening may well be compensated by the greater probability of strong union.

#### Transverse Fractures.

Owing to the minimal binding effect of the soft tissues in the transverse, this fracture is very apt to become angulated during the stages when the callus is still pliable, and, if undetected, to ossify with that deformity. It is essential, therefore, in this type of case to watch continually until solidification is complete, that gravity or muscle pull or weight-bearing does not deform the fractured limb. It is in the final weeks of treatment when the surgeon is lulled into a feeling that all is well and that vigilance may be relaxed, that bowing occurs and a bad end result follows.

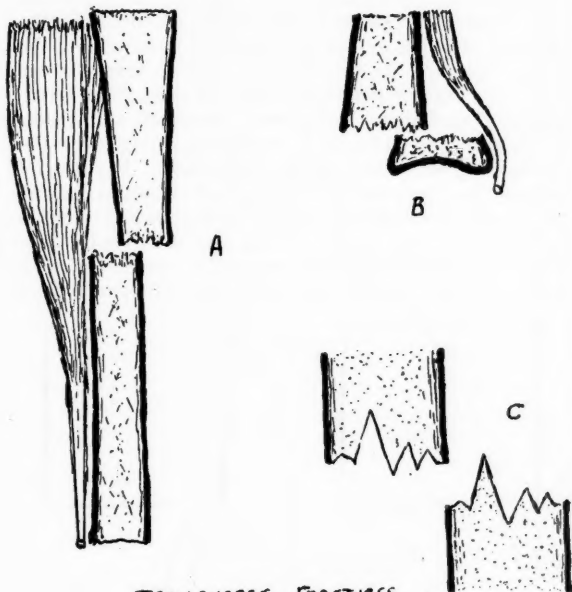
For far too long time has been the standard by which the degree of union of a fracture has been judged. The text books state, and medical men in general accept it, that in six weeks union will have occurred in a particular bone. Consequently retentive apparatus is removed and watchfulness relaxed and in many cases disaster follows.

Physical tests alone will reveal the true state of affairs. The time at which stiffening occurs will serve as a guide to the time at which union will be complete. The next test is the resistance of the repair to muscle pull. The extension apparatus only is removed and the union is subjected to the stress of muscle contraction for a period of forty-eight hours. If the alignment remains unaltered by this test, all retentive apparatus is removed and gravity allowed to exert its full sway on the junction for several days. If still no bowing occurs, weight-bearing is permitted, limited at first and watched over some days, unrestricted later and followed for some weeks.

If at any test alignment is observed to have altered, the putty-like callus is slowly moulded by continuous appropriate strain until the deformity is corrected. Only when the union of a fracture has satisfactorily responded to these tests, should the patient be allowed to escape from the observation of the medical attendant.

If the face of a transverse fracture presented a perfectly smooth surface, it would be a comparatively simple matter to reduce a displaced fragment. Unfortunately this aspect exhibits a number of irregular spikes of bone. Consequently it is necessary, as reference to Figure II, C, will show, to overstretch the limb, not merely the bones, but the muscular and fibrous structures as well, at least

by an amount equal to the length of the longest spike. In many cases it is impossible by manipulation alone accurately to replace the fragments. In the middle of the shaft (Figure II, A) exactness is not essential; as long as half of the corresponding surfaces are in apposition and the normal alignment of the limb is maintained, the functional result will be a good one. But the more closely the fracture site approaches the adjacent articulation (Figure II, B), the more insistent becomes the need for accurate replacement.



TRANSVERSE FRACTURES

FIGURE II.

Out in the open road of the mid-shaft displacement of fragments has but little effect in deflecting the pull of muscles (Figure II, A), but close to the joint in the bottle neck of the tendon traffic even a minor displacement considerably interferes with muscle functions and joint movements (Figure II, B) and anatomical reposition becomes of paramount importance. In this area the bone is close to the surface and it is possible through a small incision and with little disturbance of structures to insert a thin smooth-bladed instrument between the bone ends and by a combination of leverage and manipulation to skid the fragments into position. Once they are replaced, no traction and very little retentive apparatus are required, as the risk of recurrence is very small.

#### Parallel Bones.

If two rods of equal length are bound together at their ends, as illustrated in the diagram (Figure III), it is impossible after breaking one rod to shorten it by overlap unless its fellow is broken also or is torn from its bindings at one end.

In both the leg and the forearm are two bones lying more or less parallel and fastened together at each end. If in a skiagram the fragments of a fractured tibia are seen overlapping by more than is caused by mere alteration of alignment, then the fibula must be fractured also—it may be six

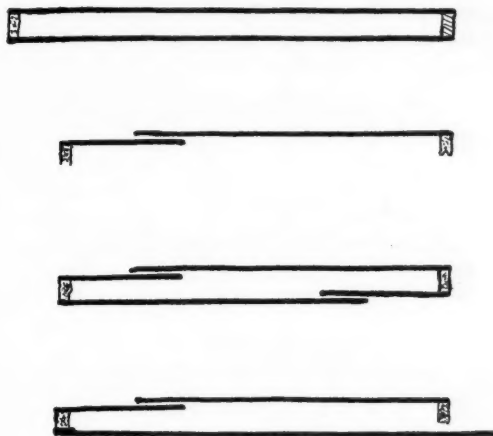


FIGURE III.

inches away. Or if X ray examination reveals an overlapping fracture of the ulna, the radius necessarily is broken, or as more frequently happens, is dislocated from its upper attachment to the ulna (Figure IV). Failure to appreciate this simple principle in mechanics, leaving unsuspected an accompanying fracture or dislocation, may lead to a serious permanent disability.

The association of anterior dislocation of the head of the radius with fracture in the upper half of the ulna is not uncommon. It appears to be produced as the result of a force applied to the palm with the elbow semiflexed, as in a head-on collision whilst gripping the steering wheel of a motor car, the violence, being transmitted along the radius, causes the head to slip forwards and upwards from the capitellum of the humerus; the ulna then having to bear all the strain, breaks obliquely in the upper half.

Plating the ulna seems to be the simplest way of maintaining the necessary extension and treatment in the fully flexed position, that of preventing the biceps from reproducing the radial dislocation.

#### Circles of Bone.

When a wooden hoop is compressed, it assumes the form of an ellipse, the apices of which are in a state of severe strain. As the pressure continues, the hoop gives way at one point and if the stress ceases at this moment, the result will be a crack in the wood with no deformity. But if the fracturing force continues, the hoop becomes severed, the ends overlap and the strain now increasing on the opposite side, this breaks also at one or other point (Figure V).

The mandibular arch may be regarded as constituting with the base of the skull a circle of bone which is subject, when injured, to similar stresses and to fracture in the same way as the wooden hoop. Thus one may expect and does find that the jaw may show (i) a simple crack without displacement, (ii) two such fissured fractures occurring in combination as body and opposite angle, body and opposite neck, angle and angle or both necks.

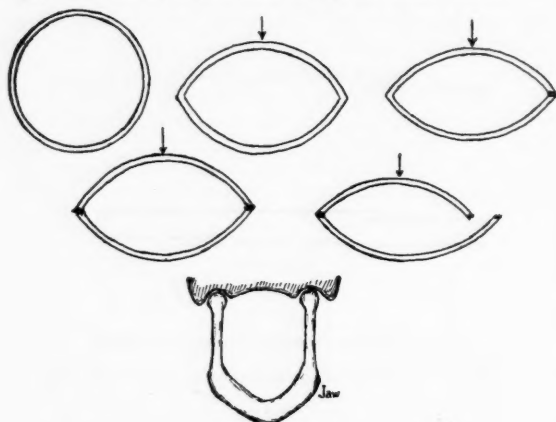


FIGURE V.

When a fracture shows marked displacement, it is most likely for the reasons given that there is a fracture on the opposite side. I am of opinion that bilateral fracture occurs in more than half the cases of fractured mandible. It is therefore necessary in the examination of a fractured jaw to have skiagrams showing the whole extent of each side of the mandible from condyle to canine area, as well as an antero-posterior view.

Another circle of bone is that presented by the pelvic girdle and this is subject to the same laws in regard to injury as is the wooden hoop. It is most frequently fractured through the pubic rami and if there is any deformity at this site, then there must be injury also to the articulations or to some other part of the bony circle. Frequently there is a fissured fracture running through the foramina of the sacrum.

#### Back Seat Fracture.

What may aptly be named a back seat fracture occurs as the result of a motor car collision following which the patient, apparently undisturbed and not having left the back seat, suffers a fracture of the pelvis. The mode of production is as follows: The momentum having shot the passenger forward so that the knee strikes the back of the front seat (and a contusion in this area is one of the diagnostic signs) an axial thrust is transmitted backwards along the line of the femur to the acetabulum. Occasionally the posterior lip is snapped off and a posterior dislocation of the hip results; but more often the force is communicated to the pelvis which fractures through the pubic rami and sacral foramina, with frequent involvement of the pelvic venous plexus and urinary tract.

Three such fractures in my experience have proved fatal from vascular and vesical complications.

#### Ankle Fractures.

Injury to the ankle joint may arise in two ways, depending on whether the foot is wrenched medially or laterally, and so there arise two distinct classes of fracture which may be described as: (i) Adduction fractures in which the whole foot is forcibly adducted and the ankle "turned out," (ii) abduction fracture in which the reverse occurs and the ankle is "turned in."

In an excellent account of fracture of the lower leg and ankle Corlette<sup>(1)</sup> has drawn attention to the confusion which exists as a result of the use of the name "Potts." It would lead to a clearer conception of what happens and what is needed in treatment, if the name Potts were dropped and terms abduction and adduction retained, indicating the mode of production of the fracture. Figure IV represents a portion of bone or of material having an internal structure or grain somewhat similar to bone such as wood. From the main mass juts down a laterally placed projection representing a tuberosity or styloid or malleolar process. If a

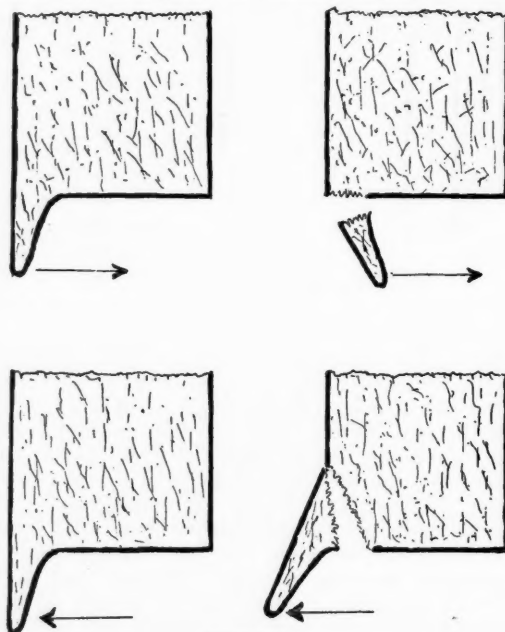


FIGURE VI.

force is applied to the apex of the process so as to pull it against the main mass, the tip of the process will be torn off or it will break midway, or as is most usual, will snap off at its junction to the main mass which is not involved. This is what happens to the styloid process of the ulna in a Colles's fracture, which is an abduction fracture of the wrist, and to the medial malleolus in an abduction fracture of the ankle.

If, on the other hand, the direction of the force applied to the process is away from the main mass



or, in other words, the projection is pushed off, the process itself does not break, but a crack occurs more or less obliquely up the main mass and the process and a laminated portion of the mass are split off. This is what happens when in an injury at the shoulder the greater tuberosity of the humerus is pushed off by collision with the acromion; in dislocation of the elbow, when the coronoid process of the ulna is forced off by contact with the lower humerus; when the lateral condyle of the tibia is split off by the femoral condyle as the leg is forcibly abducted at the knee; and in adduction fracture of the ankle where the medial malleolus is split off by means of the talus (Figure IX).

The lateral malleolus is not subject to the same mechanical laws as is the medial, because it is really a prolongation of the main shaft and not a projection from it.

When a person jumps or falls from a height, instinctively the fore part of the foot is dropped in preparation for the landing, and if in running or walking the ankle is turned, it occurs in the foremost foot at the moment when the body weight is first placed on it and before it has been dorsiflexed to a right angle. Injuries of the ankle are thus frequently associated with a degree of plantar flexion of the foot. As a result the talus is thrust upwards and backwards against the posterior lip of the concave tibial socket which is forced off (Figure VII). The split in the lower

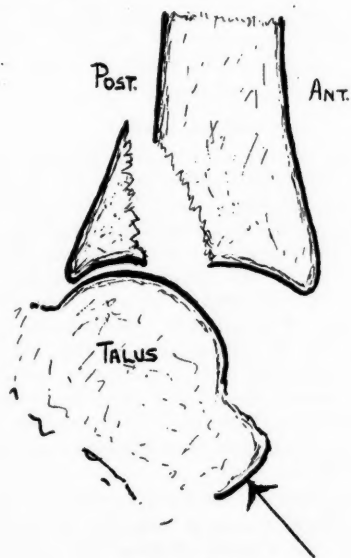


FIGURE VII.

tibia in adduction fracture does not therefore run fore and aft, but diagonally from in front of the medial malleolus backwards and outwards, so that the detached fragment, consisting of medial malleolus and post-articular lip, is forced inward and backward and the foot going with it is dislocated posteriorly (Figure VIII, 3, 4).

#### Adduction Injuries.

Forcible adduction of the foot may result in the following injuries (Figure VIII).

1. The lateral ligaments may be torn and no damage occur on the medial side beyond bruising of the talus against the malleolus. Owing to a degree of plantar flexion it is the anterior and middle fasciculi of the ligament which are injured, furnishing the ordinary sprained ankle (Figure VIII, 1).

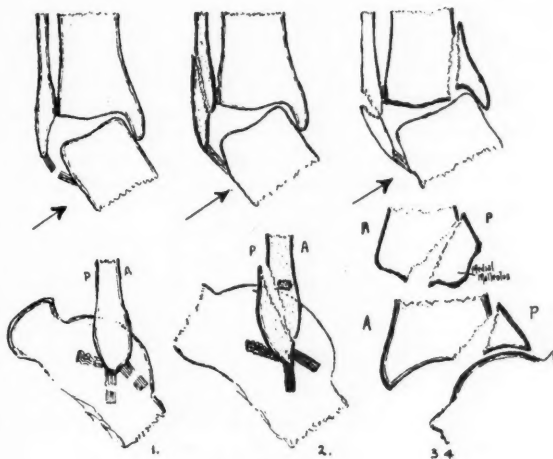


FIGURE VIII.

2. The ligament may hold and the lateral malleolus give way. Because of the plantar flexion the anterior and middle ligaments lever the malleolus inwards and forwards with the tibio-fibular fixation as its fulcrum. The malleolus cracks obliquely from above and behind downwards and forwards. This constitutes the commonest fracture of the ankle (Figure VIII, 2).

As these two types of injury to the ankle have been produced by a combination of adduction and plantar flexion, they are to be reduced and maintained in a position of abduction and dorsiflexion so as to relax the damaged ligamentous and bony structures.

3. The lateral ligaments may tear and the foot being forced still further into adduction, the talus pushes off the medial malleolus, posterior articular lip and portion of the lower tibial shaft in one fragment with resultant posterior dislocation of the foot (Figure VIII, 3, 4).

4. The lateral ligaments may hold and the lateral malleolus fracture at its base; and the adducting violence, continuing as in the third type of injury, results in fracture of both bones with posterior dislocation of the foot (Figures VIII, 3, 4; IX; X).

In the case of the last two injuries the deformity is severe and must be accurately reduced and maintained in position until repair is well advanced, otherwise there results a serious permanent disability.

With the knee in acute flexion the displacement is generally easily corrected by a combination of abduction and traction and lifting forwards of the foot. Then, as the knee is slowly extended and the powerful calf muscles tautened, the talus skids off the remaining portion of the tibial articular surface and the posterior dislocation recurs (Figure VI).

This difficulty may be overcome in various ways. The simplest method is to correct the displacement in the knee flexed position and to maintain the limb in the corrected position by a plaster slab, poroplastic or other retentive apparatus, and, still keeping up acute flexion of the knee, to leg-ropo the foot to a waist band. The powerful calf muscles are thus out-manceuvred, their spasm and pull are nullified and the greatest cause of recurrence of the dislocation disappears. Held in their zero position for ten to fourteen days, it is some time before these muscles regain their activity, and at this stage the knee may be gradually extended.

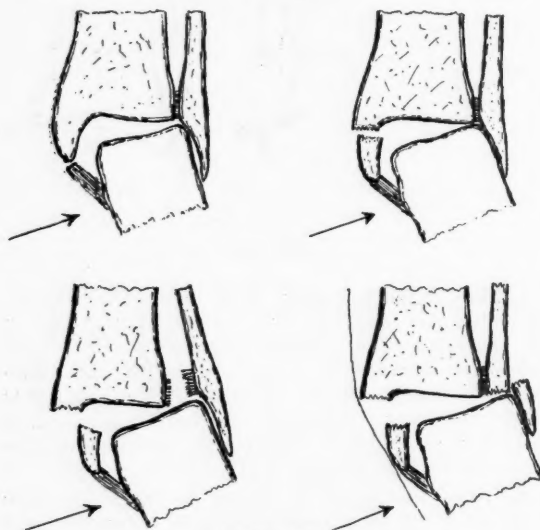


FIGURE XI.

#### Abduction Injuries.

When the foot is violently abducted, the strain first falls on the exceedingly strong medial or deltoid ligament which seldom gives way (Figure XII). Much more often the medial malleolus fails to withstand the pull and breaks at its tip or in its length or commonly snaps off at the joint level (Figure XIII). In any case the talus is forced outwards and impinges on the lateral malleolus. The strain now falls on the tibio-fibular interosseous ligaments. If this tears, the fibula, and with it the foot, is forced further out and the so-called Dupuytren's fracture is the result (Figure XIV). If the interosseous ligament holds, the fibula must go and it does so at the base of the lateral malleolus or above the tibio-fibular junction (Figures XV and XVI).

In all varieties there is an outward slide of the talus and foot in relation to the leg, and this must

be corrected at all costs. As the causative violence has been an abducting force, so reduction must be brought about by adduction of the foot. The adduction must be effective and be maintained until repair is so far advanced that the foot, when free, remains in that position; and during convalescence and the early days of weight-bearing the foot must be continually watched to see that it does not again take up the position of abduction.

One of the most frequent causes of after-pain and disability in ankle fractures is a laterally displaced talus and foot; because the body weight no longer falls through the centre of the ankle and foot, but is aligned internal to this, all the medial structures are in a condition of strain. The person with pigeon toes is sure and sound of foot. It is the patient with the weak inrolled ankle and abducted and everted foot, the misnamed flat foot, who is disabled by pain. Mismanagement of an abduction fracture will place the sufferer in the latter class. The fracture may become compound in either of these types. In the abduction form, where the medial malleolus is snapped off as the foot is forced further out, the skin becomes tightly stretched over the broken edge of the lower part of the tibia and eventually tears in a horizontal manner and directly into the ankle joint.

The adduction fracture is less frequently compound; in the case of the lateral ligaments tearing, the unbroken lateral malleolus bursts through the integuments as the foot is pushed inwards, backwards and upwards and in this manner the fibula and fractured lower part of the tibia, stripped of soft tissues, may present through a wound on the dorsolateral surface of the foot.

#### Upper Limb.

By reason of its very much greater extent of surface in the wrist joint, the lower part of the radius far outweighs the ulna in mechanical importance. Falls on the hand are practically equivalent to falls on the lower part of the radius. The radius bears the brunt of the violence in such injuries and consequently it is broken much more frequently than the ulna. The bones of the forearm are bound together by the interosseous ligament, the fibres of which run obliquely down from the radius to the ulna. The strain of upward thrusts on the radius is thus transmitted by these ligamentous fibres to the ulna. The ulna, in other words, is a secondary or support bone.

A similar mechanical state is present in the leg where the fibula forms a support or stay for the tibia. And it is worthy of note that these support bones which break after the main bone has fractured, generally unite first. The tibia and the radius are usually the site of non-union.

The majority of injuries to the upper limb result from falls on to the outstretched hand. The rigid structures of the limb are in this form of accident subjected to two forces, one an upward thrust from the resistant earth, the other a downward strain exerted by the body weight and transmitted from

the shoulder girdle. It will be obvious from a study of the photographs (Figure XVII) that the effect of the opposed forces will vary, depending upon the angle at which the outstretched arm meets the earth. For example, when the angle is one of extreme abduction, the upward thrust of the hand may act on a long lever with its fulcrum about the acromion and result in dislocation of the shoulder, or on a short lever with the tip of the olecranon as its fulcrum, and produce a backward dislocation of the elbow (Figure XVII).

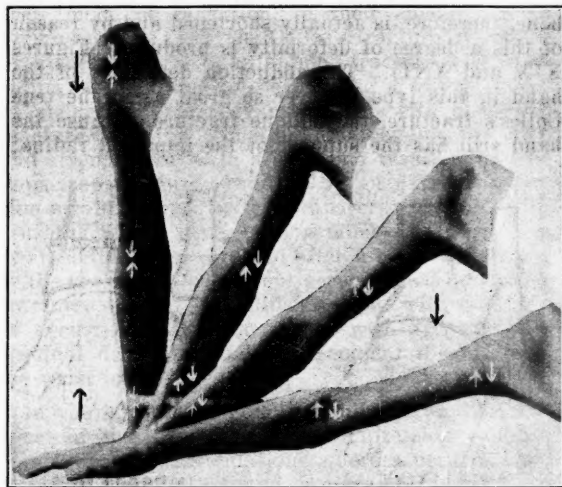


FIGURE XVII.

When the shoulder is immediately vertical to the hand, the forces will be exactly opposed along the axis of the limb and the effect will be a compression strain on all the bones and joints, an attempt to "concertina" the limb from end to end (Figure XVIII). In the presence of oblique force the strain falls chiefly at one portion of the limb, whereas in this type of compression force no part of the limb can escape the strain and consequently the lesions may be multiple. It is not unusual to find associated with sprains of the wrist, a crack in the radial head or other injury about the elbow and often with an impacted fracture of the lower radius there is damage to the articular cartilage of the shoulder.

A complaint of pain or tenderness in some other part of the arm, when a patient is suffering from a specific injury is too often disregarded or dismissed as being of reflex origin. But the character of the compression strain with its possibilities of multiple lesions should compel examination not merely of the obvious fracture, but of all the structures. It is the limb that is injured, not one particular segment of it. The unsuspected and untreated lesion gives rise to serious trouble to all parties concerned.

Between the two extremes of full abduction and the vertical position the arm may form any angle with the ground (Figure XVII). As the degree of

the angle is altered, so will be varied the resultant of the two opposing forces. The resultant will impose on the rigid structure of the limb a shearing strain, more or less oblique, according to the inclination of the arm. The shearing strain will tend to produce in the forearm and arm oblique fractures which in general will run from the flexor surface upwards and backwards to the extensor surface. In this manner there may occur in the forearm a short oblique or almost transverse fracture of both bones, usually involving the middle or lower thirds and accompanied by a degree of overlap. The problem in these circumstances is to maintain the length and to prevent any interference with pronation and supination.



FIGURE XVIII.

The mechanism whereby the radius revolves around the ulna so as to permit of rotation of the hand through almost  $180^\circ$  is a beautiful example of design and is dependent to a large extent on the normal outward bowing of the radius. When this bone is fractured in the mid-shaft, the broken ends are apt to fall in toward the ulna, the normal bow is lost and pronation and supination limited in range. Again, in transverse fracture, if the broken ends be visualized as clock faces, the corresponding hours may not be in proper relation, thus causing a rotatory deformity which will also diminish the range of pronation and supination. So in fracture of both bones of the forearm the difficulties of preventing the shortening and the rotatory deformity and of preserving the radial bow by manipulation and external apparatus alone, are so great as to justify operation by incision on the ulna and radial borders of the forearm and internal fixation of the fragments in their proper relation.

#### Colles's Fracture.

The shearing strain on the lower part of the radius produces a common arm fracture, the Colles's fracture, the line of which runs more or less obliquely upwards from flexor to extensor aspect (Figures XIX and XX). As soon as the radius breaks, the hand loses its main support and, swing-



ing on its attachments to the ulna, it and the lower fragment are forced into abduction. The strain now falls on the ulnar side of the wrist joint and the styloid process is snapped off as the hand and lower part of the radius go still further into abduction. The line of fracture is oblique and clean, so that the fragments slide easily on each other and the displacement is severe. The deformity is readily corrected by the opposing force of adduction or ulnar flexion, but is very apt to recur as the result of spasmodic contraction of the powerful forearm muscles, the majority of which act directly or indirectly on the lower radial fragment.

Reduction is obtained by adduction, that is, ulnar flexion, ulnar attachments of the wrist being used as a fulcrum. It must be maintained by that position until union is sufficiently strong to withstand the deforming action of the flexor muscles. The persistence of any degree of the abduction deformity leads to an alteration in the direction and mechanical efficiency of all tendons passing over the lower end of the radius—a condition which gives rise to a weak and painful wrist. The majority of patients presenting themselves for disability following a Colles's fracture show a persistent abduction deformity of the hand—the fracture has in most instances been completely reduced at the outset, but the deformity has recurred during the first week of treatment and has been allowed to remain unrecognized.

The cock-up position or dorsiflexion of the hand has come to be regarded as the panacea for all the ills that may afflict the wrist and its environs. For every joint which, as a result of some lesion, must necessarily become ankylosed, there is a posture which will prove best for the future efficiency of the limb. This ultimate stiffening of the joint should be anticipated and the optimum position secured at the outset of treatment. For an arthritis of the wrist there is no doubt that the cock-up position is the best, but that it should be used in cases where a freely mobile joint is to be expected or for fractures not involving the joint is very open to criticism. Indeed, in the Colles's fracture it tends to increase or at least to maintain the dorsal displacement of the lower fragment. If the hand is placed in palmar flexion the tautened extensor tendons will help to replace the dorsally displaced lower fragment, whilst the powerful flexors, being relaxed and placed at a mechanical disadvantage, have their tendency to reproduce the deformity considerably curbed. The position to be maintained in a Colles's fracture is full adduction with semi-flexion of the wrist. This is most readily achieved by the use of a plaster slab splint, moulded in the form of a gutter to the flexor surface and ulnar border and extending from the transverse palmar crease to just below the elbow.

#### Compression Fractures.

The effect of the vertical or compression force acting along the axis of the limb varies with age. In older people whose bones are more brittle, the

bone will become crushed and the force still acting in the same line will drive one fragment into the other and produce the impacted fracture of the lower end of the radius or of the surgical neck of the humerus.

When the neck of the humerus is impacted, there ensues very little interference with the functions of the shoulder joint or with the adjacent tendons. Such a condition does not call for disimpaction and may safely be left. In the case of the impacted fracture of the lower end of the radius cancellous bone is crushed and rendered more compact; the bone, therefore, is actually shortened and by reason of this a degree of deformity is produced (Figures XIX and XXI). The abduction deformity of the hand in this type is never so great as in the true Colles's fracture, the oblique fracture, because the hand still has the support of the impacted radius;

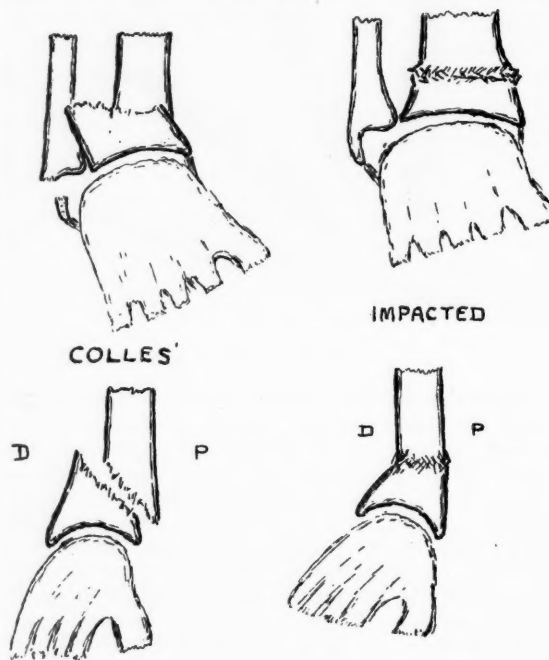


FIGURE XIX.

and for the same reason it is not accompanied by snapping of the ulnar styloid process. Sometimes the extensor aspect of the bone is impacted and suffers to a greater degree than the flexor, and as a result there is angulation of the lower fragment with backward tilting of the lower articular surface, preventing full flexion at the wrist (Figures XIX and XXII). The displacements of abduction and backward tilting interfere with the normal direction of tendon travel and with the range of movement of the wrist and they should in general be corrected. Reduction is difficult and is brought about by manipulation and traction; occasionally operative intervention is indicated. Traction is maintained by means of adducted position of the hand, the hand being used as a lever and the ulna as a fulcrum to



draw the radial fragment into position; semiflexion of the wrist will tend to correct the backward tilting of the fragment. As there has been an incrusting of bone, so when the fracture has been disimpacted there will be a gap between the fragments. For this reason the deformity is very apt to recur and also union will be delayed, so that the management of this fracture calls for a longer period of fixation and of watchfulness.

Fractures of the lower third of the radius are therefore divisible into two classes, differing in their mode of causation, in their effects and in their prognosis: (a) The Colles's fracture, produced by a shearing strain, an oblique fracture with overriding of the fragments, exhibiting a severe degree of abduction deformity associated with a styloid fracture, easy of reduction and capable of early and strong union with a good end result. (b) The impaction fracture produced by an axial compression strain, a more or less transverse fracture with crushing of the bone, with a much smaller degree of abduction deformity and often a backward tilting of the articular surface, not accompanied by styloid fracture, reduced with difficulty and with tendency to recurrence, slow to unite, with frequently a residual deformity and a permanent disability of the wrist joint.

In youth, with its greater resilience of bone, the effect of the axial thrust or compression is often manifest at the articulations where a fissured fracture into the joint is found. An example is one form of chauffeur's fracture produced in cranking a motor car when at the moment of backfire the body weight is thrusting vertically down the straightened arm. The articular surface of the radius receives the full force of the blow and a star fracture results, comprising fissures radiating from the joint in one or more directions, but with practically no displacement. An injury such as this, involving a fracture into any joint, should be regarded less as a fracture of bone and more as a damage to the joint and should be treated on similar lines to an infective arthritis, that is, by a greater degree and longer duration of fixation than would be given to a broken shaft. Accordingly, the fissured fracture into the wrist joint should be treated in the cock-up position in anticipation of an adhesive arthritis and should be given complete rest.

Another compression fracture is seen at the elbow, when as a result of a vertical fall on to the hand the axial thrust along the radius may jolt the concave head against the under-surface of the humeral condyle and result in splitting of the head. If the fracture is a crack without displacement, it should be treated as already indicated by plaster fixation of the elbow at a right angle. If the fragments of the head are split off and separated, these will seriously interfere with the movements of the joint. This type is best dealt with by excision of the fractured head a week after injury. Almost perfect movement and function of the elbow are thus obtained.

#### Olecranon and Patella.

In fractures of the olecranon and patella accurate restoration of the normal articular relations of the fragments is essential to unimpaired function of the joint. These bones at the extremes of movement of the elbow and knee are not in such intimate contact with their respective condyles as in a position midway between full flexion and extension. Consequently, it appears that a better reposition of the fragments will be obtained by moulding them to the articular ends of the humerus and femur in the position of semiflexion. This position entails a greater tension on the muscles which produced or are maintaining the separation of the fragments than does the position of extension and so imposes a greater strain on the material used to bind the broken bones.

In certain of the methods used for repairing these fractures the wire is made to traverse badly damaged bone in some part of its course (Figure XXIII, A). Such a procedure is mechanically unsound because the wire speedily cuts through traumatized bone as the result of pressure and quickly becomes loose and ineffective. A safer anchorage for the binding material is obtained at the site where tendon is inserted into bone. Separated patella fragments may be securely bound by an encircling suture of wire, chromicized gut or other material threaded through the tendons (Figure XXIII, B). And in the case of the olecranon a wire passed through the triceps tendon and through a hole drilled in the dense bone of the ulnar ridge, will hold the fragments firmly in apposition (Figure XXIII, C).

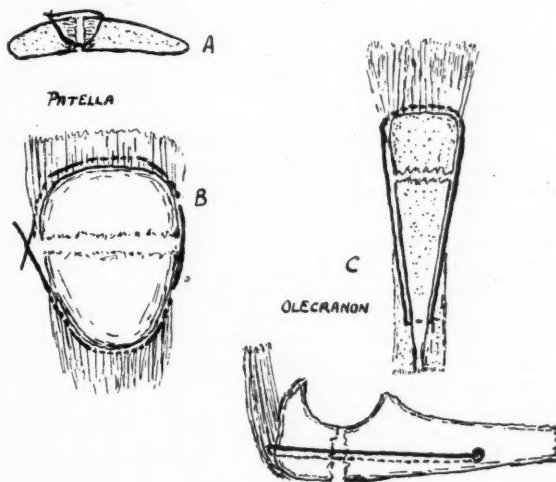


FIGURE XXIII.

#### Fracture of the Tibial Spine.

Extension of the knee is checked chiefly by the powerful crucial ligaments. Over-extension of the joint imposes a strain which results in damage to the ligaments or to their bony attachments. If the crucial ligaments give way, there follows one form of dislocation of the knee. Damage to their femoral

attachments may give rise to a condition known as *osteocondritis dissecans*. When the tibial attachments are affected the result is fracture of the tibial spine.

A detailed account of the accident which has resulted in fracture of the tibial spine will frequently reveal that the injury was caused by hyperextension of the knee, and the fact that X ray examination will often show a lamina of bone lifted from the articular plateau of the tibia, suggests that overstrain of the crucials is the mechanical principle involved. These injuries are usually treated by fixation of the knee on a back splint in the fully extended position, a position which places the damaged structures on tension, and is therefore incorrect. Relaxation should be afforded by semi-flexion of the knee and as the lesion is intra-articular, the period of fixation should be prolonged and when weight-bearing is allowed, extension of the joint should be checked by the use of a Marsh's cage splint.

#### Spiral Fracture of the Tibia.

The spiral fracture of the tibia is the result of a torsion strain on the bone which may be applied in two ways, namely: (i) The body and limb are twisted whilst the foot is fixed, or (ii) the foot is twisted whilst the body and limb are prevented from turning.

When a person is thrown or jumps from a moving vehicle and lands on one foot, that foot becomes fixed whilst the spinning momentum of the body has the effect of twisting the leg. On the other hand, when the body is fixed on one foot, a twist is imparted to the other as it is placed and skids on an inclined plane in the act of walking. In either case the tibia is twisted to its breaking point and a spiral fracture results, usually in the middle and lower thirds of the leg.

In the great majority of cases the violence does not cease immediately the tibia breaks, but continues and the more flexible fibula now has to bear the whole strain of the torsion. It eventually fractures and always at its upper extremity, the neck breaking in a spiral manner or the head becoming comminuted (Figure XXIV). Hence a spiral fracture of the lower part of the tibia is always associated with fracture of the fibula, fifteen centimetres (six inches) away. The fracture is one in which it is exceedingly difficult to obtain accurate replacement of the fragments. The bone ends are always sharp and pointed and the corkscrew action of the breaking force tends to drive them into the surrounding soft parts. The shape of the tibial fragments presents another obstacle to reduction, whilst the comminution and displacement of the fibula contribute also to the difficulties.

As the violence has been of a twisting nature, so reduction should be attempted by combination of untwisting and traction in order to overcome the effect of the torsion on the fibula and soft tissues and to disengage the tibial fragments.

#### Reference.

<sup>1</sup> C. E. Corlette: "Malleolar Fractures (sometimes called Potts's Fracture) and Supramalleolar Fractures," THE MEDICAL JOURNAL OF AUSTRALIA, July 16, 1927, page 76.

#### THE SKIN TOLERANCE FOR PRESSURE AND PRESSURE SORES.

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It is well known that the effect of an agency upon living tissue varies greatly with its concentration or intensity. Drugs which in excess cause death of tissue, when acting in small amounts stimulate growth. Bacterial irritation of minor degree calls forth a reaction on the part of the tissues, yet the same irritant in greater concentration destroys the living cells. Pressure as an influence is no exception to the rule. In this paper the reaction of the skin, and more particularly the normal skin, to pressure influences will be discussed.

Two factors, the intensity and the duration of the pressure influence, must be considered. The skin is capable of withstanding very high pressure for a short period or even for a great number of short periods, if there are intervals of freedom from excessive strain. In standing the skin of the sole is called upon to withstand compression which is enormously in excess of the blood pressure. Obviously the vessels of the sole must be temporarily obliterated as channels and, if the pressure were maintained for a long time, the skin would certainly die. Of course it never is so maintained, and at each step or movement of the foot the skin is flushed with blood. As the intensity factor is increased, there comes a point above which pressure acting even for a brief moment destroys the tissue by actually disintegrating the cell arrangement. A heavy blow from a hammer acts in this way.

Of more importance is the effect of the second factor, duration. Sores are produced as a rule by quite moderate pressure acting continuously over a long period. It is of first importance to know just how much pressure the skin can tolerate indefinitely and how pressure in excess of this may be avoided.

The normal systolic blood pressure of about 120 millimetres of mercury corresponds roughly to 2.3 pounds to the square inch. The skin tolerance for pressure is likely to be well below this. In order to ascertain the normal skin pressure tolerance a small rubber bag with a tube attached was made and the bag was bandaged on to the dorsum of a boy's foot. Precautions were taken to avoid the possibility of circular constriction by first fitting a plaster sole slab which projected beyond the margins of the foot and insured that a large part of the skin of the dorsum should be free from pressure. A single layer of lint was placed between the skin and the bag to provide for aeration and drying of the part. The tube was fitted with a funnel which could be raised or lowered by the boy. The

bag, tube and funnel were filled with water. The boy was told to raise the funnel slowly, inch by inch, until he experienced discomfort. He found that he could stand a pressure of about thirty-nine inches for days without experiencing pain. On inspection, the skin was apparently unaffected. The skin pressure tolerance over the dorsum of this boy's foot was thus about 1.5 pounds to the square inch. In other parts it is probably more or less the same, but it is likely that deficiency in the nerve or blood supply would lower the figure. A similar experiment on a man with a systolic blood pressure of 110 millimetres of mercury gave a figure of 1.25 pounds to the square inch. The boy's blood pressure was 126 millimetres of mercury.

It is quite possible that a series of observations would show that the skin pressure tolerance varies slightly with the blood pressure.

#### Pressure Sores.

It is clear that pressure of more than 1.5 pounds to the square inch over a long period is likely to cause death of skin, even if this were previously quite healthy. Important questions come up for consideration. How does it come about in practice that the toleration point is exceeded with resulting sores; and what precautions should be taken to insure that this may not happen?

The surface area of a man whose height is five feet eight inches and weight 150 pounds, is about 2,790 square inches. If only one-fifth of this surface were available for weight bearing in the recumbent position, then, supposing that pressure were evenly distributed, it would amount to less than one-third of a pound to the square inch. This pressure, far from being harmful, will tend, by supporting the tissues, much as an Unna's stocking does, to preserve the well-being of the part. In practice, however, in an ordinary bed the weight of the body falls heavily on certain points whose combined area is small, lightly on other parts and not at all on some places. It is obvious that metal splints of the Thomas type, of which the surface area is quite inadequate for weight-bearing and which fit very imperfectly at the best, so tending further to limit the effective surface, cannot be expected to do anything else but produce pressure sores unless, as happens in practice, the patient is largely raised from the splint by means of pillows tucked between and around the bars.

Sores commonly occur over those parts of the bony skeleton which closely approach the skin surface, that is, the heel, the sacrum, the posterior superior iliac spines, the spines of the vertebrae and so on. The skin at these points is always in contact with the bed surface, and hence is called upon to bear not only the weight of the structures immediately above, but also that of the intervening parts which are not adequately supported. Further, there is a tendency for the bulky soft parts, such as the buttocks and calves, to deform and thus evade the responsibility of carrying the weight of the tissues directly above. There is no escape for the thin soft

parts over the skeletal prominences already mentioned. This settling of the skeleton will be referred to again. It is clear, then, in answer to the first question, that high pressure is developed over prominent points because other parts of the surface between and around these are not taking their fair share of the burden.

The answer to the second question as postulated above is obvious from what has been said in regard to the first. It is this. The whole of the skin surface available for weight-bearing must be used, thus insuring that the pressure will not rise to anything like the tolerance point at any one place. Special care must be taken that the skin over skeletal prominences is subjected to rather less strain than elsewhere.

A skilled nurse who has the above principles in mind will be able, by the careful arrangement of pillows which have a certain fluidity, to insure that the prominent danger points are not called upon to bear excessive strain, and in the case of most bedridden patients who can be rolled over, this is all that is required. When, however, it is inadvisable to move the patient often, or when there is more than usual danger of sores, as in the case of helpless patients with poor tissues or subnormal sensation and so on, then it is necessary to resort to measures designed to distribute the pressure more evenly than can be done by the nurse.

Two curiously different apparatus are available: first, a water bed and, secondly, a plaster bed, the one fluid, the other quite rigid. Each has its advantages and disadvantages. The surface of the water bed adapts itself well to a fairly large skin area, and is easy, once set up, to manage. It does not provide immobilization, which is sometimes desirable. Whilst pressure is very well distributed over a large area of body surface, the prominent points still bear more than their fair share on account of the fact that the envelope interferes with the perfect fluid action of the bed. The plaster bed, when well made, is in contact with a larger surface of the trunk than is the water bed, as it supports the oblique postero-lateral contours of the buttocks, flanks, thorax, neck and, if necessary, head. The skin over the prominent danger points is subjected to less pressure than elsewhere, provision for this having been made during the construction of the plaster. Immobilization is very good. It should be noted that the hard surface does not *per se* damage the skin. No thick material, especially cotton wool, should be introduced between plaster and patient, as it is liable to wrinkle or ruck up and produce high pressure points. A single layer of silk or other thin material is an advantage, as it helps to obviate the risk of rubbing the skin when this moves on the plaster. It is by no means unusual for patients to lie in a plaster bed for months without any inspection of or attention to the back, and sores do not develop. There are, of course, patients whose tissues are so abnormal that sores develop in spite of all precautions, but only as a rule amongst the moribund.



Sometimes trouble arises at the point where the lower edge of the plaster presses on the skin of the sacral area or buttocks. This may be due to a badly shaped edge, but most often results from the fact that the lower limbs are not supported throughout their length so as to transmit their weight directly to the underlying bed surface. Each limb thus resembles an arch supported at two points, the hip and the heel; and as in an arch the weight is concentrated at the springing stones, so a proportion of the down thrust of the limb will fall on the skin of the sacral area, which is thus called upon to bear a double burden. This difficulty is best overcome by the careful use of pillows introduced so as to insure that each part of the leg receives adequate direct support. In some cases the plaster is extended so as to sustain more or less of each or either limb.

Reference has been made to what may be termed settling of the skeleton. When, for instance, a plaster bed is moulded on the patient lying prone, an almost perfectly fitting shell is obtained, which might be expected to distribute pressure evenly over the skin surface in question when the position is reversed so that the body is supported on the plaster. But the body consists of soft parts and the skeleton. This latter because of its own weight and that of attached soft parts lying above it tends to sink into the underlying soft parts, much as would a leaden core in a ball of cotton wool. Suppose that the skeleton approaches nearer to the under-surface of the body by so little as one-eighth of an inch. This would have small effect on the skin at those points where an inch or two of muscle separates it from the overlying bone; but where this distance is, say, three-eighths of an inch or less, as over the sacrum or vertebral spines, the effect may be disastrous. The tissues between the two hard structures, bone and plaster, are rendered anæmic and soon slough. To avoid this trouble pads of wool about one-quarter of an inch in thickness are placed over all bony prominences prior to construction of the plaster. When these pads are removed, shallow depressions are left in the bed surface for the reception of the danger points.

#### Beneficial Effects of Moderate Pressure.

When a part of the body is put at rest in a dependent position, it swells. Apparently the return of lymph is imperfect. The tissues become bathed in stagnant fluids and soon show the effects of impaired nutrition. Varicose ulcers might more descriptively be called stagnation ulcers. It is common knowledge that slight pressure, such as that exerted by a well applied Unna's stocking, causes the œdema to disappear and immeasurably improves the state of the part, so that ulceration is averted if impending, cured if already present. It is reasonable to argue that the static œdema of the subcutaneous soft parts of the back seen in bedridden patients lowers the resistance of the tissues and predisposes to ulceration, and that moderate pressure designed to prevent stagnation would be a powerful factor in the prevention and treatment of

decubitus ulcers. For this reason it has been urged in this paper that the danger points over the skeletal prominences should not be entirely freed from pressure, but that an endeavour should be made to arrange matters so that they shall take rather less than other points. Hence the provision in the plaster bed of only very shallow depressions for their reception. The annular air cushion so much favoured in the nursing of bedridden patients would appear to favour the production of a state of stagnation opposite its central aperture. It is probable that moderate, possibly intermittent, pressure of, say, eighteen inches of water on and around an ulcerated area would favourably influence the state of the part.

#### SOME ASPECTS OF BRONCHIAL ASTHMA AND ALLIED CONDITIONS.<sup>1</sup>

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I thank you for the honour you have done me in asking me to discuss some aspects of bronchial asthma and allied conditions.

#### Bronchial Asthma.

In the first place, when a history of asthma is obtained, you will recall the necessity of excluding nephritis, aneurysm, mediastinal neoplasm, heart failure, Hodgkin's disease and pulmonary tuberculosis as possible causes of the asthmatic paroxysm. I make it a routine to carry out an X ray examination of the chest in all cases of supposed asthma, because it may be difficult to exclude some of the above conditions clinically, and in addition valuable information may be obtained as to changes in the lung fields, signs of pleurisy, cardiac enlargement and other departures from the normal.

When possible, the sputum should be examined macroscopically and microscopically, including an examination for tubercle bacilli. If it is decided that the condition is one of true bronchial asthma and not due to any of the above conditions, an investigation as to the cause should be made.

A careful history is necessary to ascertain the date of the first attack, whether preceded by any illness such as coryza, bronchitis, pneumonia *et cetera*, the frequency and duration of the paroxysms, and whether they occur in summer or winter or all the year round.

Colds, coughs, bronchitis or pneumonia are frequent precursors of asthma and in such cases chronic bronchitis, bronchiectasis, infection of the sinuses of the skull, or naso-pharyngeal infection may be maintaining the disability. If no infective cause can be found, inquiry must be made as to the occupation, environment, association with horses, cats, fowls and other animals, the bedding, dusts, pollen of flowers and, lastly, foods.

<sup>1</sup> Read at a meeting of the Eastern Suburbs Medical Association, New South Wales, July, 1930.



In my experience in this country asthma due to pollens is less common than has been reported elsewhere; occasionally a definite horse or cat asthma is found, or a relationship to a particular dust or one food substance is proved. Eggs, wheat and some fruits are rare causes. So are nuts, orris root *et cetera*. If no obvious cause has been found, it may be necessary to employ skin tests for those substances which cannot be otherwise excluded. In my hands this method has proved helpful only in rare instances, but in any case it is important to go over the possible causes again and again in the hope of finding the main cause. When any evidence of infection in the nose, throat or bronchi is found, an X ray examination of the sinuses is advisable. Naturally, a complete examination of the patient is necessary to determine any nasal obstruction, increased blood pressure, emphysema or possible focus of irritation outside the respiratory tract.

I will not describe the asthma paroxysm at length; you are very familiar with that and with the recorded pathology of it.

Asthma may occur at any age and in young children it may be difficult to be certain whether the condition described is asthma or whether it is due to laryngeal spasm; I recall one case in a child with extraordinary difficulty in expiration, so that I thought there must be a foreign body in the trachea, in which relief followed rest in bed and the injection of adrenalin.

You also are aware how frequently signs of asthma are wanting between the paroxysms and how important it is not to mistake a history of pulmonary oedema or cardiac asthma for one of true bronchial asthma. When an examination is made at the time of an attack the diagnosis is usually obvious enough. I have been deceived by the history in cases of aneurysm and of cardiac and renal asthma more than once.

As you know, the prognosis is very variable; some patients appear to have only one attack in a life time, but as a rule the tendency is for the condition to recur and often in a way which is still inexplicable.

In regard to the difficulty of determining the cause, I may mention that Van Leeuwen's latest method of diagnosis is to place his patients in a special pollen and dust proof building, drawing in air from a height of twenty or thirty metres. Those patients who continue to have asthma in this building, he regards as non-allergic; in those who do not, he searches for a protein to which the individuals are sensitive.

#### *Treatment.*

I think any fresh treatment often relieves asthma for a time at any rate; I still believe that lobelia and iodides are very valuable drugs in asthma; often the combination of alkalis with iodides causes easier expectoration.

For the paroxysm ephedrine relieves some; adrenalin I still have greater faith in; but I think

it is a good practice to avoid these drugs, and also morphine, if at all possible.

The avoidance of a heavy night meal often prevents or diminishes the nocturnal attacks and an easily digestible diet is essential, as is attention to the bowels. I often use rhubarb or colocynth and mercury.

As regards specific treatment, avoidance of any discoverable cause is obvious.

In bacterial asthma treatment of the naso-pharynx, antral disease or chronic bronchitis must be carried out, and the use of vaccines from the naso-pharynx or sputum is often followed by improvement, if the patients' statements are any guide. In some cases desensitization against pollens or moulds may be helpful. Of non-specific treatment, peptone injections given intravenously, as advocated by Auld and others, give much relief for a time in some cases.

#### *Hay Fever.*

In regard to hay fever; this is generally a seasonal condition, coming on most commonly between September and March, while grasses and flowers are pollinating.

For proper investigation by skin tests a knowledge of the time of pollination of the common grasses and flowers is necessary. Winter grass pollinates earlier than most other grasses and seems to coincide with the time of onset of the majority of cases of hay fever in Sydney.

Cape weed pollinates later, and is now widespread in New South Wales. Testing for pollen sensitivity is tedious and often it is difficult to be sure of the causative pollen. House dust gives a positive reaction in some cases, perhaps due to moulds. My own experience of these methods of diagnosis and subsequent treatment by pollen extracts has not been universally successful.

Hay fever varies so much in severity from year to year that it may be difficult to ascertain the value of treatment. It is possible to aggravate the condition by so-called desensitization.

Locally cocaine 1% to 4% gives most relief; ephedrine and adrenalin solutions benefit some patients. Some authors advocate closing up the room so as to exclude pollens or dust, or a change of locality is advised to avoid the causative pollen. The former often causes a feeling of suffocation and the latter is scarcely applicable to any large section of the community.

Measures to improve the general health are necessary in some cases, and nervous subjects benefit by the administration of bromides and arsenic.

#### *Giant Urticaria.*

Giant urticaria (angioneurotic oedema) is the most dramatic of the conditions allied to hay fever and asthma. It may be due to bacteria, pollens, foods or dusts. I have seen patients cured by tonsillectomy and by treatment of infected sinuses, and I have seen very marked skin reactions to the

dust of oats; this patient was particularly affected by going into the room where horse feed was kept. Some patients are relieved by ephedrine or adrenalin and a mercurial pill; and alkaline mixtures or a change of environment have been associated with recovery in a number of cases in my experience.

In this condition careful investigation is of the utmost importance and, if negative results are obtained, start again at the beginning. It would take too long to give a list of possible causes; these can be found in text books on the subject. Elimination of possible food causes from the diet one by one may be necessary. I have had no experience of desensitization in this condition and would be inclined to proceed warily with this method.

### Reports of Cases.

#### A DANGEROUS TYPHOID CARRIER.

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AND

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AFTER a community has provided itself with a good water supply and a good sewerage system, the most important steps have been taken in the campaign against typhoid fever. There is, however, in all modern communities a residual amount of typhoid, of which it is extremely difficult to get rid. When we consider that there are probably only about one or two carriers per thousand in an average city population, the hunting for these is something like hunting for the proverbial needle in a haystack. This makes it all the more important that the occurrence of sporadic cases which may indicate the whereabouts of the carrier should be used to the utmost for his detection. The three cases of which we give brief notes, indicated very clearly the whereabouts of one such carrier.

#### Case I.

E.W., aged eleven years, was admitted to the Alfred Hospital on June 26, 1928, apparently suffering from pneumonia. Before admission he had been ill for five days, during which time he suffered from fever, loss of appetite, drowsiness, vomiting, coughing, epistaxis and delirium. During the two days immediately preceding admission he had vomited everything that he had eaten.

Physical examination on admission revealed signs of consolidation at the base of the right lung. No enlargement of the spleen or liver could be detected. On July 3, 1928, that is to say, seven days after admission and twelve days from the commencement of the illness, a blood culture was taken and from it *Bacillus typhosus* was isolated. On July 6, 1928, the patient died.

During the ten days he was in hospital his temperature averaged about 40° C. (104° F.) (see Chart I).

#### Post Mortem Findings.

The base of the right lung was found to be consolidated and in a condition of grey hepatization. Ulceration of Peyer's patches was found in the small intestine. The mesenteric glands were enlarged and inflamed.

#### Comment.

If this case had been observed in private practice without blood culture and without post mortem examination it is highly probable that it would have been regarded as a

pneumonia only and its typhoidal character overlooked. Price,<sup>(1)</sup> in referring to the difficulty of diagnosis of typhoid fever in childhood, writes: "The disease does occur in childhood and is then likely to be overlooked."

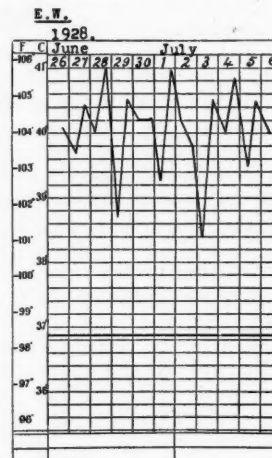


CHART I.

#### Case II.

On March 31, 1930, R.W., aged seven years and eight months, brother of the first patient, was admitted to the Alfred Hospital with a provisional diagnosis of pneumonia. He had taken ill three days before admission with vomiting, sweating, headache and stiffness of the neck. Immediately before these symptoms arose the patient had had one tooth extracted.

On admission an examination of the abdomen revealed no enlargement of the spleen or other physical sign of interest. There was some dullness of percussion at the apex of the right lung. On April 5, 1930, bacteriological examination of the urine gave negative results.

On April 8, 1930, a blood culture was carried out with negative results. On April 11, 1930, agglutination was attempted against the typhoidal organisms, *Brucella abortus*, *Micrococcus melitensis* and *Bacillus proteus*, also with negative results. On April 15, 1930, the motions were suggestive of typhoid fever. On April 17, 1930, a second blood culture also gave negative results.

On April 19, 1930, no splenic enlargement could be detected, epistaxis was present, but the motions were still suggestive of typhoid fever. On April 22, 1930, agglutination tests were carried out with the same organisms as had been used before, again with negative results. On April 23, 1930, samples of faeces and urine were examined bacteriologically, using MacConkey's medium, but no enteric organisms could be isolated. On April 24, 1930, slight melena was present. The temperature curve is illustrated on the accompanying Chart II.

It is an unusual but well known type of enteric fever. Such more or less complete intermissions of fever are described by various authors as occurring in typhoid. On April 29, 1930, the patient died.

#### Post Mortem Findings.

Typhoidal ulceration of the terminal three feet of the ileum, together with ulceration of the caecum and ascending colon, was found. There was evidence of recent intestinal hæmorrhage and pelvic suppuration. *Bacillus typhosus* was recovered by culture from the gall bladder.

#### Comment.

This case is interesting because of the completely negative character of all the laboratory findings during life, and illustrates again the difficulty of diagnosing typhoid fever in children.

Whether the recrudescence of fever should be looked upon as indicative of a relapse or not must be left an

ILLUSTRATIONS TO THE ARTICLE BY DR. FAY MACLURE.



FIGURE IV.  
Showing overlap of ulna with dislocation  
of the radius.



FIGURE X.  
Showing fracture of both malleoli and of posterior  
articular portion of tibia with backward dislocation of  
the foot.



FIGURE IX.  
Adduction injury, showing fracture  
of medial and lateral malleoli.



FIGURE XII.  
Abduction injury, showing tearing of medial  
ligament, outward slide of foot, and fracture  
of lateral malleolus.



FIGURE XIII.  
Abduction injury, showing fracture of medial  
malleolus at joint level.

ILLUSTRATIONS TO THE ARTICLE BY DR. FAY MACLURE.



FIGURE XXIV.  
Showing spiral fracture  
of the tibia with fracture  
of neck of the fibula.

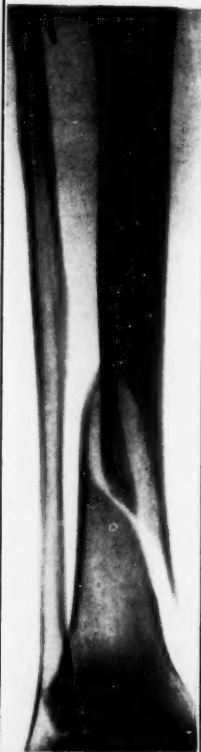


FIGURE XIV.  
Abduction injury, showing fracture of medial  
malleolus at joint level, outward slide of  
foot, tearing of tibio-fibula ligament, and  
fracture of the fibula.



FIGURE XVI.  
Abduction injury, showing fracture of both  
malleoli at joint level with extreme lateral  
displacement of foot.



FIGURE XXI.  
Showing impacted fracture of the lower  
end of the radius.



FIGURE XV.  
Abduction injury, showing fracture  
of both malleoli with lateral out-  
ward displacement of foot.



FIGURE XX.  
Colles fracture, oblique from the palmar  
upwards to the dorsal surface.



FIGURE XXII.  
Showing impacted fracture of the radius  
with backward tilting of the lower frag-  
ment. Note the transverse line of fracture.



open question. If during the first and second spells of fever we had been able to cultivate the typhoid bacillus from the blood on each occasion, we might reasonably have regarded the second period of fever as a relapse, but the precise significance of these two fevers must be left in doubt. The *post mortem* findings, however, are convincing that the patient actually died of typhoid.

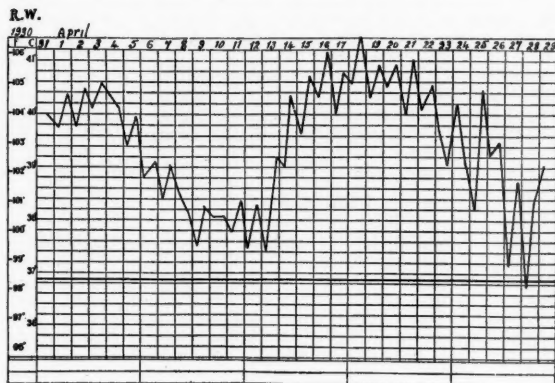


CHART II.

An interesting point about this case was that intestinal hæmorrhage had occurred which is unusual in children. The negative agglutination results in similar cases have been believed to be due to the extreme severity of the attack. Whether this was the explanation here or whether it was due to some peculiarity of the antigen infecting the patient or used in the agglutination test, it is not possible to say.

We would suggest, however, that where the physician is satisfied that a patient who is showing no antibody response, is nevertheless suffering from typhoid fever, then a large dose of antityphoid serum might be administered with advantage.

#### Case III.

B.W., a female, aged five years, was admitted to the Alfred Hospital on August 19, 1930, with a temperature of 40.2° C. (104.4° F.), a pulse rate of 140 and a respiratory rate of 40.

The family history given was of great interest, namely, that the father and mother and four children were alive and well, but two children (previously described in this paper) had died of typhoid, one about five months ago and one about two years ago. This third patient was ill for four days before coming to the hospital, suffering from pains in the legs, fever, sweating and delirium.

Four days after admission and eight days from the commencement of the fever a blood culture was taken and gave negative results. Two days later agglutination tests were carried out and gave the results set out in the accompanying table.

Organisms.	Patient's Serum in Dilutions of				
	1/20	1/40	1/80	1/160	1/320
<i>Bacillus typhosus</i> ..	+++	+++	+	—	—
<i>Bacillus paratyphosus</i> A ..	—	—	—	—	—
<i>Bacillus paratyphosus</i> B ..	—	—	—	—	—
<i>Brucella abortus</i> ..	—	—	—	—	—
<i>Micrococcus melitensis</i> ..	—	—	—	—	—
<i>Bacillus proteus</i> X19 ..	—	—	—	—	—

Four days later *Bacillus typhosus* was isolated from the faeces.

The accompanying Chart III shows that the temperature of the patient rapidly fell and became normal.

#### Comment.

Typhoid fever in childhood is alleged to be of slight severity. The three cases that occurred in this family have

all been severe and two of them ended fatally. It seems not unlikely that some of the severe cases of typhoid in young people are diagnosed as pneumonia and this would give a false impression of the severity of the disease.

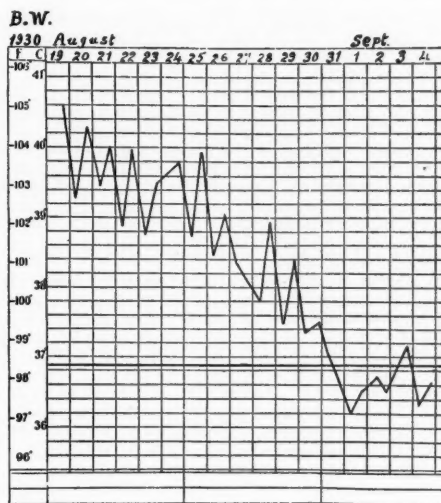


CHART III.

#### Detection of the Carrier.

When the laboratory became aware that this was the third attack in the family, samples of faeces and urine were obtained from every member of the household (seven). The examination of these samples revealed the presence of *Bacillus typhosus* in a boarder who came to the house a little over two years previously and about five weeks before the first child died. After residing with the family for five months he left them for a year and about six weeks after his return the second child died, since when he has continued to reside with the family. The results of the bacteriological examinations, therefore, and the relationship of the movements of the boarder to the deaths of the first two children are convincing evidence of the boarder's responsibility for producing the disease. When the former cases were notified, an inspector and medical officer of health visited the home on each occasion, but according to the mother no faeces or urine was sent to any laboratory for examination in an endeavour to find the carrier. No vaccination was carried out to protect the residents. Even now, after the detection of the carrier, it appears that no suitable provision for isolation is available for him, although he has been clearly demonstrated to be a menace to his neighbours.

The Australian medical literature is not rich in references to dangerous typhoid carriers detected in Australia, but an article by C. T. Ch. de Crespigny<sup>(2)</sup> gives details of a nurse who was a carrier and whose return home from hospital after an attack of typhoid was attended by the occurrence of no less than nine cases of typhoid in her family, three of the cases being fatal. If such patients are not going to be isolated, prophylactic inoculation should at least be administered to all close contacts of the carrier.

#### Acknowledgement.

Our thanks are due to Dr. F. Kingsley Norris for permission to use the notes of the cases.

#### References.

- <sup>(1)</sup> F. W. Price: "Practice of Medicine," 1926, page 62.
- <sup>(2)</sup> C. T. Ch. de Crespigny: "Notes of a Typhoid Carrier," THE MEDICAL JOURNAL OF AUSTRALIA, July 30, 1921, page 79.

## Reviews.

### PROGRESS IN PÆDIATRICS.

As with the previous edition, the second edition of "Recent Advances in Diseases of Children,"<sup>1</sup> by Pearson and Wylie, is rather in the form of a text book, in which, while dealing with the subject mainly from the clinical aspect and in a form and manner met with in everyday practice, the authors have correlated and incorporated such important recent additions to knowledge as in their opinion have a bearing on the study of disease in childhood.

The most important alterations are to be found in the chapters on encephalo-myelitis and cerebral diplegia, on chronic infection of tonsils and adenoids, on bronchiectasis, thoracic tuberculosis and asthma, on chronic abdominal conditions, congenital syphilis and on skin tests and immunization.

Much emphasis is laid on the necessity for a complete and accurate history of both child and parents, and differentiation is made into two main types of child, lymphatic and neuro-arthritis, with the suggestion that the former is a hypocalcemic condition, requiring acid forming foods, and that the latter is a hypercalcemic state.

In the chapters on feeding, the uses and the indications for the administration of foods containing adequate supplies of vitamins and salts are well defined. The diets for older children are given in detail, though, as usual, various breads and patent foods are mentioned which are unknown in this country. There is an excellent account of the cerebro-spinal fluid in health and disease. The X ray plates are excellent and typical of their kind. There is a short but useful chapter on immunization and immunity.

### AUSTRALIAN LITERATURE.

MR. H. M. GREEN'S "Outline of Australian Literature" is the first work of its kind to appear in Australia, and it has certainly been undertaken by a most competent authority.<sup>2</sup> The book is very welcome and we must feel particularly grateful to the author for piloting us through waters hitherto uncharted, for classifying and criticizing the material involved, and perhaps for reminding some Australians that an Australian literature does exist. We are told in the preface that this book is merely an outline, to be followed later by a more amplified history of the literature. But even as it stands the "Outline" is most helpful and informative. It deals with all literature that genuinely portrays Australian conditions and characteristics, whether or not the author be Australian by birth. The literature, from which is excluded all that is not "creative", is divided into five periods. A final chapter is devoted to drama and essays—forms of literary expression but little exploited in Australia. Mr. Green might be criticized, when his work begins so far back as 1795, for devoting so much space to writers since 1914. Yet since the volume and perhaps the worth of literary matter have gained in later years, this seeming disproportion may be justified.

Particular prominence is given to Lawson who is considered as the most representative of Australian writers, to Brennan in whom the intellectual and imaginative elements are strongest, to Brereton, O'Dowd, McCrae, Slessor and Henry Handel Richardson. The multitude of lesser writers is not easy to focus. Mr. Green states that some of them are not so well known as they should be. But even with the kindest feeling, not many readers will find fault with the public taste that has allowed some of those included in the book to remain in congenial obscurity. On the other hand, we are surprised to find missing such writers as David Blair, John Sandes, Frank

Myers and Davidson Simmonds, who would certainly seem far more worthy of mention. Mr. Green has, however, a keen sense of literary values and has dealt admirably with the problems that faced him; his criticisms are for the most part discriminating and sympathetic. Though it is notoriously difficult to estimate impartially what is near at hand, he shows an authoritative detachment and penetration that, while making us feel that we can accept his judgements, yet stimulate a desire to see for ourselves.

The author of the "Outline" is himself a prominent figure in Australian literature. Known to us chiefly through his verse, Mr. Green possesses the genuine poetic gift and the "Book of Beauty," with its fairy atmosphere, subtlety of rhythm and its loveliness of imagery, is perhaps unsurpassed in the literature of this country.

### THE SLIT LAMP.

THE second edition of Koby's "Slit Lamp Microscopy of the Living Eye" is virtually a new book.<sup>1</sup> The first edition was a good book, the second is an extremely good book. It is now an imposing volume of over three hundred and fifty pages, having grown beyond a third of the size of the first edition. This fact alone serves to demonstrate the growing importance of the slit lamp in everyday clinical work. It is no longer a refinement or a luxury, it is just as much a necessity to the ophthalmologist as a trowel to a bricklayer. It is not to be supposed that the clinician who gets along without the assistance of the slit lamp is doing bad work; rather is it that he is denying himself the help of the greatest clinical aid science, since the perfection of the ophthalmoscope, has yet put at his disposal, and without the knowledge made accessible by a slit lamp examination of the anterior portion of the globe, a modern day opinion cannot be given upon such conditions as the pathological pigmentation of the cornea in the entities known as Fleischer's ring, the deposit accompanying Wilson's disease, or hepato-lenticular degeneration, and the Kruckenberg's spindle. Nor can the earliest evidence of iritis, irido-cyclitis or sympathetic ophthalmia be detected. Many other conditions could be enumerated in which the slit lamp has opened up new aspects and even modified the terminology employed.

The book is an excellent tutor for the specialist who in Australia needs must rely upon himself for instruction in the use of the apparatus, and with its aid no one need shrink from the task of becoming self taught. The only criticism that can be made is that there is no index to the one hundred and four illustrations throughout the text.

### MASSAGE AND EXERCISES.

In "Mechano-Therapy" Dr. Mary Rees Mulliner has satisfactorily carried out her intention of supplying the needs of students.<sup>2</sup>

The procedures of massage and the physiological effects are described in the fullest detail and in simple language. The student is told not only what are the appropriate exercises for a special condition, but is shown how to "make a prescription," indicating how to arrange the "day's order" so that the best results can be obtained.

Special reference should be made to the chapter on neuromuscular economy which might be read with profit by others than massage students.

The influence of posture in the causation and treatment of many pathological conditions is emphasized.

On the whole, the book can be recommended to all students and practitioners of mechano-therapy as a very complete and lucid exposition of the subject.

<sup>1</sup> "Recent Advances in Diseases of Children," by Wilfred J. Pearson, D.S.O., M.C., D.M., F.R.C.P., and W. G. Wylie, M.D., M.R.C.P.; Second Edition, 1930. London: J. & A. Churchill. Demy 8vo., pp. 558, with 20 plates and 34 text-figures. Price: 15s. net.

<sup>2</sup> "An Outline of Australian Literature," by H. M. Green; 1930, Sydney: Whitcombe and Tombs. Crown 8vo., pp. 288.

<sup>1</sup> "Slit-Lamp Microscopy of the Living Eye, Early Diagnosis and Symptomatology of Affections of the Anterior Segment of the Eye," by F. E. Koby, translated by Charles Gouldon, O.B.E., F.R.C.S., and Clara Lomas Harris, M.B., Second Edition; 1930. London: J. and A. Churchill. Demy 8vo., pp. 376, with 104 illustrations. Price: 15s. net.

<sup>2</sup> "Mechano-Therapy: A Text-Book for Students," by Mary Rees Mulliner, M.D.; 1929. Philadelphia: Lea and Febiger. Post 8vo., pp. 265, with illustrations. Price: \$2.75 net.

## The Medical Journal of Australia

SATURDAY, NOVEMBER 29, 1930.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

### MEDICAL HISTORY.

THE study of history means much more than the collection of facts—of happenings, of dates and of the lives of persons. Facts are, of course, the essential ground work, the foundation on which the superstructure is built. When concrete facts have been ascertained, they may be arranged in their proper time sequence and according to their causation. After searching criticism as to their relative value, and then only, may the facts be submitted to deductive reasoning. It is quite clear that if facts are to be interpreted in the light of their arrangement and of criticism, great caution is necessary. The student who would carry his historical research to this point, must shed all personal bias and be free from preconceived ideas. In proportion as he brings cold logic and calm judgement to bear on his problems will his study become scientific. Thus the methods used in historical research are essentially similar to those pertaining to research in general and the history of medicine makes exactly the same demands on its students as does the history of any other subject.

Medical history may be regarded in several ways. No writer or research worker dreams of undertaking the investigation of a subject without inquiry as to what has already been done in the field of his

choice. His research into the past is a necessity to him, for it is not only unnecessary, but foolish, to spend time and energy in going over ground that has already been traversed, unless it be by way of confirmation of results in a matter that is yet in the stage of speculation. Most serious workers recognize the need for research of this kind. Medical history may also be studied from the literary and the human points of view and it may be sought as a source of inspiration. It is these aspects on which we wish to lay stress. Probably no subject in the whole range of human endeavour is so full of human interest, no subject holds such romance, no subject is more replete with stories of self-sacrifice and devotion to an ideal and no subject can give greater encouragement and impetus to renewed effort. The story of Hunter, of Pasteur, of Lister and of the other giants in medicine will never fail to rouse the interest and thrill the imagination of even the dullest. Such achievements as the discovery of the life history of the malarial parasite and the conquest of yellow fever in Central America will always compel attention. But men of lesser calibre have also left "on the sands of time" footsteps that may be followed with advantage. This is true of every age and every clime—it is true of Australia, young though she be.

Though in recent years increased attention has been given in Australia to medical history, there is still room for improvement. The medical practitioners of Australia have reason to be grateful to several of their number. The late Norman J. Dunlop gave of his best to historical research for many years and his work takes its place in the foremost rank. Dr. E. Sandford Jackson has published in this journal important researches into the records of the early days in Brisbane. The names of Dr. A. A. Lendon, Dr. John MacPherson and of others could be added to the list. We have drawn attention to the difficulty of historical research and to the qualities necessary to one who would undertake it. There must be many in Australia possessed of these qualities and of a love of literature for its own sake. A large field remains to be explored. There is a Section of Medical History and Literature of the Australasian Medical Congress (British Medical Association) and the New South Wales



Branch has a section devoted to this subject. From both these sources interesting contributions to Australian and general medical history have emanated. It is also to be noted that the Victorian Branch has appointed a small subcommittee to collect an early medical history of Victoria, and those possessed of information in regard to early Victorian practitioners have been asked to communicate with Dr. G. T. Howard and Dr. Felix Meyer. It is hoped that the response to this appeal will be spontaneous and generous. We venture the suggestion that each of the Branches in Australia should put aside one meeting in the year for the reception of contributions dealing with historical and literary subjects. This would be a stimulus to members gifted in this direction. It would also tend to increase the value of the contributions and stimulate the interest shown in the Section of Medical Literature and History of the Congress when it meets every three years.

### Current Comment.

#### PNEUMONIA AND PNEUMOCOCCI.

THE bacteriology of pneumonia is not even yet clearly established. It has been shown by Somerville that pure cultures of pneumococci can be obtained from examples of bronchopneumonia and that in lobar pneumonia mixed organisms occur. Liston examined forty instances of fatal bronchopneumonia and obtained Pfeiffer's bacillus in all but two. In thirty-three this organism was obtained in pure culture. Next in frequency were streptococci. Pneumococci were relatively rare. R. Miller suggests that Pfeiffer's bacillus may break down resistance and allow ready access to other infective organisms. T. Smith has stated that the *Streptococcus pneumoniae* (pneumococcus) is not infrequently the cause of disease in animals. Balzy has isolated from three instances of septicæmia in calves a strain which proved serologically identical with a human strain. E. Palier recently advanced the theory that the house mouse is the chief agent in the transmission of pneumococcal diseases. A series of articles has recently been contributed by American authors concerning pneumococci in relation to the human subject and experimental animals.<sup>1</sup>

E. G. Stillman states that rabbits are very susceptible to infection by Type I pneumococcus. As regards Type II, he made experiments with three

strains. One strain was converted into the "R" form by repeated transfers in Type II anti-pneumococcus serum. The changes from "S" to "R" form are accompanied by loss of capsule, of type specificity and of virulence for mice. This strain is termed pneumococcus IIR and is avirulent to rabbits. The original pneumococcus Type II strain is termed pneumococcus Type II (SAv). An intraperitoneal injection of 0.1 cubic centimetre of a broth culture of this stock strain is necessary to kill rabbits. It is rendered rabbit-virulent by rapid passage. This virulent strain, called pneumococcus Type II (Sv), regularly kills rabbits when injected intraperitoneally in doses of 0.00001 cubic centimetre. The susceptibility of rabbits to inhalations of pneumococci varies in direct proportion to the virulence of the organism for rabbits. If rabbits are exposed to a pneumococcus spray irrespective of the virulence of the organisms, the bacteria readily penetrate into the lower respiratory tract. If they are exposed to a spray of avirulent degenerated "R" forms, septicæmia does not occur and pneumococci are rarely recovered from the liver, kidney or spleen. When rabbits are exposed to a spray of slightly virulent Type II (SAv), a non-fatal septicæmia may occur and pneumococci may be recovered from the liver, kidneys and spleen. When rabbits are exposed to virulent Type II (Sv), septicæmia may occur which may terminate fatally and pneumococci may be recovered from the organs mentioned. Rabbits and mice may acquire a high degree of immunity following repeated inhalations of living pneumococci. After repeated inhalations of degenerated non-virulent Type II "R," no type specific antibodies can be demonstrated in the serum of rabbits. Following repeated inhalations of slightly virulent Type II (SAv) only protective antibodies can be seen in their serum. After repeated inhalations of virulent Type II (Sv) agglutinins and protective antibodies are found in their serum. After repeated exposures of rabbits to inhalations of pneumococci, the type specific response, evidenced by type specific protective antibodies and agglutinins varies in direct proportion to the virulence of the culture used. The strain of Type I used produced high mortality among rabbits. Many of the survivors had agglutinins and protective antibodies in their serum. Following inhalation, virulent Type I generally disappears from the lungs of rabbits within a few hours, to reappear later in certain instances in which general septicæmia occurs. Some may recover, but rabbits are very susceptible to infection by pneumococcus Type I.

John F. Enders presents evidence for the existence of a substance distinct from the specific carbohydrate in the autolytic products of pneumococcus Type I. This substance reacts specifically by precipitating homologous antiserum, which occurs naturally without antibody against the specific carbohydrate or has been deprived of that antibody artificially. In guinea-pigs passively sensitized with such antisera the homologous autolysate containing

<sup>1</sup> The Journal of Experimental Medicine, August 1, 1930.



the substance alone produces typical lethal anaphylactic shock. In weakly alkaline solution the substance is destroyed by boiling. In weakly acid solution it resists a temperature of 100° C. for at least half an hour. It is resistant to peptic digestion. It is possibly, in chemical nature, a haptene. That man is fairly resistant to the pneumococci is evidenced by the comparative rarity of contact infection amongst nurses and others attending pneumonia patients. In such conditions pneumococci are doubtless frequently inhaled in large numbers by the contacts. When such infections occur, pneumococci of Types I and II have been those generally observed. The inference might be that these types are the most virulent for man. It is a matter of common observation that pneumococci are often identified in the sputa of persons who have not pneumonia and who, to their knowledge, have never suffered from it. Pneumococcus carriers are numerous among the population.

O. H. Robertson and M. Agnes Cornwell contribute a study of the resistance of normal human beings to recently isolated strains of pathogenic pneumococci. It has been found that the resistance of an animal to pneumococcus infection is paralleled closely by the pneumococcicidal power of the blood. When the serum and leucocytes of the rabbit were tested against pneumococci of varying rabbit virulence, the pneumococci of low virulence were destroyed in considerable numbers, while those of high virulence grew readily in the serum-leucocyte mixtures. The serum and leucocytes of young rabbits which are highly susceptible to pneumococci of low virulence for adult rabbits, possessed no pneumococcicidal action. There was a quantitative relationship between the killing dose of pneumococci and the pneumococcicidal power of the animals' blood. Experiments were made as to the pneumococcicidal action of normal human serum-leucocyte mixtures. The study gave no evidence that any one type of pneumococcus was of higher virulence for human beings than the others. The pneumococcicidal potency of pooled normal human serum and leucocytes exhibited a surprising uniformity for the several strains of pneumococci. The resistance to pneumococcal infection, especially of the lungs, depends on local and general factors in addition to the pneumococcicidal power of the blood. Probably the pneumococcicidal power of human blood indicates the human capacity to combat invasion by pneumococci in any part to which the blood has free access. In relation to the high mortality of lobar pneumonia caused by pneumococcus Type II, it was surprising to find that a group of fifteen normal persons tested showed a more constant pneumococcicidal power for this type than for other types. That this result was not to be attributed to the employment of strains of relatively low human pathogenicity was indicated by the fact that two of them were isolated from the blood in fatal cases; one infection was fulminating, the patient dying within forty-eight hours. There was, however, some evidence to suggest that the virulence of those Type II strains for

human beings may have diminished before being used in the tests with normal individuals. It is of interest that an individual should exhibit marked variations in pneumococcicidal activity for different types of the organisms. An occasional but only slight deviation was observed in cats and dogs. The reactions of the human serum-leucocyte mixtures are more comparable in this respect with those of a pneumococcus-susceptible animal, such as the rabbit, which shows marked individual fluctuations both in resistance to pneumococcus infection and in concentration of natural humoral immune bodies for different pneumococcus types. Whether or not these apparently specific reactions of the human blood elements depend on the presence of distinct opsonins for the several types of pneumococci was not determined. As a group human beings show well marked pneumococcus destroying power for all types of the pneumococci studied. Individuals, however, showed wide variations in their reactions against the different types, ranging from a marked killing effect on one type to slight or none against another. The reactions against different strains within the type often varied greatly; but this difference on the whole was less than between different types. An interpretation of these findings in the light of animal experiments in which actual determination of resistance to pneumococcus infection was made, justifies the inference that human beings in general possess a considerable natural immunity to all types of pneumococcus, but that individuals may be relatively susceptible to one or more types while resistant to others. Further, pathogenic strains of pneumococci vary greatly in their virulence for man.

#### QUINSY AND DIPHTHERIA.

THE differentiation of faucial diphtheria and quinsy is sometimes a matter of difficulty. If a wrong diagnosis of peritonsillar abscess is made, the illness becomes more acute and the patient often succumbs. J. E. Gordon and D. C. Young have reported that of forty-three patients whose diphtheritic fauces were incised in the belief that peritonsillar abscess was present, twenty-five died.<sup>1</sup> This rate, 58%, is much higher than that for all types of diphtheria. Among 3,260 cases of diphtheria there was only one instance in which peritonsillar abscess was associated with acute diphtheria. Adults are most usually concerned, because diphtheria is suspected in throat conditions in children. With a pyogenic infection high fever is more usual. Bilateral swelling of the fauces should suggest diphtheria, and unilateral swelling peritonsillar abscess. Incision of peritonsillar abscess can generally be postponed until a bacteriological examination is made. Gordon and Young make the useful suggestion that when no pus is obtained after incision of a supposed peritonsillar abscess, diphtheria antitoxin should be given.

<sup>1</sup> *Archives of Internal Medicine*, September, 1930.

## Abstracts from Current Medical Literature.

### SURGERY.

#### Elephantiasis of the Scrotum and Penis.

STUART BERGSMAN (*American Journal of Tropical Medicine*, May, 1930) describes an operation for the relief of elephantiasis of the scrotum and penis. The first incision is made from the opening of the long external urinary tract to the *glans penis*, laying open the tract. A transverse incision is then made for a distance of 3.81 centimetres on either side of the vertical incision at its upper extremity. A vertical incision is then made from each extremity of the transverse incision to the base of the penis and the flap thus formed is dissected up. The skin in this situation is usually healthy, as it has been dragged down from above by the weight of the tumour, though it may overlie elephantoid tissue from which it must be freed. The penis is freed from elephantoid tissue, the testicles and cords are freed by dissection, wrapped in hot, wet sponges and placed on the suprapubic region. An incision is then made encircling the base of the tumour, leaving a flap for the scrotum 5.08 to 7.62 centimetres in length. This skin is usually healthy also. The penis is encircled with the flap left for the purpose, the distal end of the flap being sutured to a collar of prepuce left for the purpose when the penis was dissected out. The testicles are brought down and the scrotal flap sutured. The author claims that this method utilizes healthy skin which is usually discarded in operations for the relief of scrotal elephantiasis, and obviates the necessity for undercutting the skin of the thighs for the purpose of making scrotal flaps.

#### Fat Necrosis of the Breast.

GEOFFREY HADFIELD (*The British Journal of Surgery*, April, 1930) writes about fat necrosis of the breast. The commendable modern practice of radically excising any new formation bearing a reasonable clinical likeness to malignant disease in a patient of the cancer age has brought into some prominence and provided examples of an innocent lesion of the breast, presenting itself most often in women between the fourth and fifth decades, frequently as a stony-hard tumour firmly fixed to the skin, often resembling an early cancer so closely that a wide resection of the breast has been performed. The central feature of the process is slow, aseptic saponification of neutral fat by blood and tissue lipase, analogous to pancreatic fat necrosis, but much milder in degree and independent of any direct action of pancreatic lipase. The lesion arouses suspicion chiefly by reason of its situation in the fat overlying the breast and copies the physical signs of cancer most closely in the early and middle stages of its evolution, when it

is usually stony hard. At no stage does its naked eye appearance on incision or its microscopical characters bear a close resemblance to cancer, but its histology may conceivably suggest tuberculosis. Amongst the conclusions of Lee and Adair are: (i) Traumatic fat necrosis of the breast is a definite clinical entity; (ii) it must always be included with the benign lesion of the breast; (iii) clinically it more closely resembles carcinoma of the breast than any other tumour; (iv) a distinct history of trauma to the breast and a well circumscribed mass showing a rapid increase in size, unassociated with pain and without axillary nodes that are firm, suggest the possibility of fat necrosis; (v) the diagnosis of traumatic fat necrosis of the breast by gross examination is possible. Fat necrosis of the breast is therefore by no means unique as a pathological process; its importance lies in its liability, purely due to situation, to mimic the clinical signs of mammary cancer.

#### Thoracic Surgery.

PETER WALZEL (*Wiener Medizinische Wochenschrift*, February 8, 1930) considers the development of thoracic surgery one of the greatest conquests of the post-war period and pays homage to those men who have worked so hard in this special branch of surgery, especially Sauerbruch. He discusses empyema first. The extensive thoracotomy operation with open drainage is followed by disastrous results if performed before the mediastinum is fixed, but if there are signs of increasing intrathoracic pressure with disturbance of respiration and circulation, then immediate surgical interference may be necessary, whether the empyema be either of the two chief varieties, namely, tuberculous or secondary to other infections. He regards the purpose of an operation as twofold, namely, to evacuate the fluid from the pleural cavity and to relieve the pressure which is displacing the mediastinum. The open technique must never be employed unless the mediastinum is fixed and all signs of the causal inflammation have settled down; except in empyemata following gangrene of the lung and rupture of a lung abscess, when the stinking pus and sequestra of dead lung tissue may require free and early evacuation. In all other instances closed drainage by paracentesis and various kinds of continuous suction should be tried, especially in children. Frequently the condition is cleared up in this manner, but if after some weeks the empyema is still present and drainage is becoming more difficult, he performs rib resection, packs the entire cavity with gauze for several days, and then, after its removal, promotes the expansion of the collapsed lung with breathing exercises and frequent irrigations of the cavity. A certain percentage, however, remain chronic and must be submitted to an extensive plastic operation as devised by Kirschner and

Schede, together with avulsion of the phrenic nerve. Primary tuberculous empyemata and those following artificial pneumothorax should have closed drainage as long as possible. He then discusses lung abscesses following pneumonia, septic infection elsewhere and inhalation of foreign bodies or septic material during general anaesthesia. The chief indications for surgical treatment are the duration of the illness, the degree of septic absorption and the spread of the abscess, especially if medical and postural treatment has been of no avail. Before attempting to drain a lung abscess by open operation, it is necessary that the visceral and parietal pleurae in its vicinity should be adherent if empyema is to be prevented. This can usually be determined by radiological examination. If not, the operation must be performed in stages. At the first stage the pleural space over the abscess is packed off with gauze which forms adhesions before the second stage is performed two weeks later. Sauerbruch's method of injecting paraffin extrapleurally, to press the layers together, three or four weeks prior to operation, has proved excellent, and on occasions curative, without further interference. The more conservative treatment, by artificial pneumothorax, is effective in certain instances. He regards the surgical treatment of bronchiectasis as presenting many problems, and gives a summary of current ideas concerning the aetiology, pathology and diagnosis of this distressing malady, with special reference to the taking of bronchograms after "Lipiodol" injections. For large isolated bronchiectatic cavities treatment similar to that of lung abscess may be tried. When one lobe only is affected, modified thoracoplasty, extrapleural filling and phrenic nerve avulsion have been advocated, but have only been met with indifferent success. Ligation of the main branch of the pulmonary artery to the affected lobe and lobectomy after having previously shut off the pleural cavity by adhesions in an operation consisting of three stages have been reported by Sauerbruch with success. He finds it impossible to do justice to the wonderful results obtained by surgical treatment of suitably selected patients suffering from pulmonary tuberculosis and to describe fully the various procedures adopted. As a result of close cooperation between physicians and surgeons, a definite routine has been arrived at. Artificial pneumothorax treatment is given by the physicians, but if collapse is prevented by pleural adhesions, then extrapleural thoracoplasty or extrapleural compression, with or without phrenic nerve avulsion, is undertaken by the surgeons. Among the indications for thoracoplasty he stresses a good resistance on the part of the patient, unilateral infection and a fibroid type of the tuberculosis. In such cases Sauerbruch claims 60% to 80% of cures by his operation. For the large number of persons infected unilaterally with

an exudative type of tuberculosis and whose general condition is poor, he advocates Baer's method of extrapleural compression of the lung with paraffin, especially when large isolated cavities exist. In the author's clinic in the last two years Sauerbruch's paravertebral thoracoplasty has only been performed on rare occasions. He favours Baer's extrapleural compression which has been completed with resulting clinical improvement in one hundred instances with an operative mortality of only two *per centum*. This series includes three patients who had not benefited after thoracoplasty, and many patients who were seriously ill. Operations for the removal of simple tumours and cysts of the lungs, mediastinum or chest walls may be very extensive and difficult, but offer good prospects of a cure. Primary carcinomata of the lung and bronchi present a more difficult problem. For the former excision in two stages of the affected lobe has been successfully carried out by Sauerbruch. Recurrences, however, are likely; for carcinomata of the bronchi surgery offers no prospects of relief.

#### Treatment of Mastitis.

H. KÜSTNER (*Münchener Medizinische Wochenschrift*, January 3, 1930) declares that the old classification of breast infections into "parenchymatous" and "interstitial" types has no real significance, as in every instance there is no clear line of demarcation in the breast tissue. He advocates complete rest to a breast in which there are signs of inflammation and uses ice and a binder in the early stages. If the inflammation advances, hot fomentations are substituted and when fluctuation occurs the abscess is thoroughly evacuated. In three instances when severe gangrene of the skin existed, he obtained good results from the application of the quartz lamp for one to two minutes at a distance of one metre. Six treatments were given in each instance. The good effect was considered to be due to the destruction of all organisms in the affected area as well as to a stimulatory effect on the general constitution of the patients.

#### Cancer of the Rectum and Rectal Polypi.

VICTOR SCHMIEDEN (*Deutsche Medizinische Wochenschrift*, November 29, 1929) reminds his readers of his previous contention that cancer of the rectum is always preceded by definite precancerous tissue changes, namely, the heaping up of narrow polypus cells, which occur in the varied forms of polypi of the colon and rectum, and that the prognosis of rectal polyposis has become more serious with the knowledge that a certain group of polypi will surely become carcinomatous at a certain age. He has done further work on this subject with Westhues, and in answer to his critics, especially Sauerbruch, submits further proofs of the frequency of early cancer occurring in polypi of the rectum and

colon and its appearance in multiple foci in the same segment of mucous membrane. In cancer of the rectum he has found remains of the preexisting polyposis and of all intermediate grades of development, including involvement of lymph glands in the surrounding mucosa, by means of a careful examination of serial sections. He submits a series of microphotographs showing polypi and all the degrees of cancerous changes. Some of the earliest occur at the tips of simple looking polypi, and even then the lymph glands may be already involved. Finally, whereas in other diseases rectal polypi are rarely seen, they always predominate in cancer of the rectum. He urges every surgeon to make a thorough and critical examination, both macroscopical and microscopical, of all polypoidal fields. Only in this way can his theory be finally proved.

#### Death on the Operating Table.

B. BREITNER (*Wiener Medizinische Wochenschrift*, August 9, 1930) presents a very exhaustive analysis of the causes of death on the operating table. His work is based on a study of 155 deaths occurring in 152,360 cases in Vienna. One-half of these followed ether anaesthesia and one-third chloroform. However, when the deaths were calculated on a percentage basis, the true position was noted; ether anaesthesia had been followed by a mortality of 0.04% as compared with 0.1% for chloroform. A careful review of the cases shows the importance, as predisposing causes, of conditions such as peritonitis, ileus, general sepsis, marked coronary sclerosis, cachexia, severe anaemia and fatty degeneration of the heart. A careful physical examination is especially needed with the newer forms of anaesthesia, as any pronounced organic changes are contraindications to their use.

#### Acute Obstruction of the Small Intestine.

R. V. B. SHIER (*The Canadian Medical Association Journal*, May, 1930) reports twenty-nine cases of obstruction in the small intestine occurring in his own private practice, and summarizes the collected statistics of several authors. In 1925 Soutter collected over 3,000 cases of obstruction to the intestine which occurred in seven London hospitals from 1920 to 1924 inclusive. The mortality rate of obstruction to the small intestine from all causes was 30%. Deaver states that the mortality should not exceed 10%. The toxæmia associated with obstruction of the small bowel has its origin in protein decomposition due to the action of bacteria. Death in cases of strangulation without gangrene is due to dehydration, loss of chlorides and starvation. It may be delayed by the administration of fluids and chlorides. In strangulation with gangrene death is due to toxæmia. Normal mucous membrane apparently will not absorb a lethal dose of toxin. Pain referred

to the umbilical region is the initial symptom of obstruction of the small intestine. A history of previous abdominal inflammation, operation or hernia may be obtainable. Pain due to hernia may be sensed only in the umbilical region. Visible peristalsis may be expected only in the poorly nourished. The onset of a wave of pain after abdominal palpation is highly suggestive of the condition. Acute pancreatitis is the chief pitfall in diagnosis, but here the pain is more constant and severe and is usually associated with a leucocytosis. Intravenous use of saline solution and glucose must be commenced early as a preoperative measure. Spinal anaesthesia is the author's choice. Insufficient relaxation is obtained with a gas anaesthetic. The performance of ileostomy depends on the extent of bowel filled with fluid and the degree of toxæmia. No good results from draining bowel filled with gas.

#### Surgery in Experimental Heart Disease.

WILLI FELIX (*Deutsche Medizinische Wochenschrift*, September 13, 1929) points out that the bad results obtained after operations on the heart itself or its valves in cases of heart disease led him to investigate extracardiac means of providing relief. After describing the physical forces at work in producing hypertrophy and dilatation of the heart, he defines the problems facing surgery as being to allow dilatation where it is called for and to prevent it when not wanted. He then discusses the external forces at work on the heart and selects the tension of the pericardium for investigation. He produced mitral and tricuspid valvular insufficiency in animals, then by a release of pericardial pressure by splitting open the pericardial sac, the dilatation and the signs of heart failure which had resulted, were made to disappear and efficiency restored by compensatory hypertrophy. With the other valvular lesions constant effects were not obtained because they caused hypertrophy alone. In acute and chronic arterial hypertension removal of pericardial tension caused a lowering of blood pressure, although hypertrophy and dilatation resulted. In a similar series of cases he increased the pericardial pressure by taking a tuck in the sac; and in all instances a decrease in the heart's efficiency resulted with dilatation. In certain instances, by paralysing one half of the diaphragm, this dilatation could be delayed and even prevented. He utters a warning, however, that these results may not be applicable to man. He concludes with a plea for further experimentation. Observation should be made after Sauerbruch's decompression treatment, for it lessens the thoracic pressure, fixes the chest in inspiration and assists diastolic filling. Right-sided pneumothorax therapy, which increases the pressure on the heart by decreasing the elastic pull of the lung, should also be considered.



## Special Abstract.

### NEONATAL MORTALITY.

THE new-born baby's sudden transition from a state of physiological dependence to one of physiological autonomy marks a critical period in human existence. Uncertain and unstable, even with the protection of full physiological development, life at this early stage is fragile and apt to be destroyed by the lightest pathological touch. The race is the poorer for every death of a worthy infant, and the importance of the study of neonatal mortality and its causes, many of which are preventable, cannot be too strongly emphasized. Any authoritative communication on the subject merits the earnest consideration of every medical man.

J. N. Cruickshank, sponsored by the Medical Research Council, is the author of a recent report on the causes of neonatal death.<sup>1</sup>

#### Introduction.

For the purposes of his communication Cruickshank defines the neonatal period as the first four weeks of extrauterine life.

The work on which his opinions are based, was a series of eight hundred *post mortem* examinations made by him at the Glasgow Royal Maternity and Women's Hospital. Though he admits that a full and accurate diagnosis is not always possible at autopsy, he suggests that the clinical examination of young infants is so difficult that the information gained therefrom is not nearly so reliable for statistical purposes as that obtained by *post mortem* examination.

Of the 800 deaths investigated 71% occurred during the first week, 18% during the second week, 8% during the third week and 3% during the fourth week. From the year 1925 to the year 1927 there occurred in the Glasgow Royal Maternity and Women's Hospital 6,887 live births and 474 neonatal deaths (6.8%). The percentage of deaths occurring during the first week was 76.7 and the death rate during the first week was 52.7%.

#### Classification of Causes of Neonatal Deaths.

Classified according to their causation, neonatal deaths fall into two main groups, namely, those resulting from injury or disturbance of function incurred during birth and those resulting from acquired illness or infection. In two smaller groups are listed deaths due to developmental anomalies and deformities, and deaths due to visceral thrombosis or hemorrhage resulting from maternal toxæmia. Many of these latter are actually due to asphyxia and are included under that heading in this report. In this connexion it may be noted that recent work has tended to minimize the importance of the effects of environment and maternal malnutrition on the newly-born infant. Bruce Murray found that mothers with insufficient resources bore children not appreciably inferior in development to those of mothers who were better provided. Fairbairn remarked that Bruce Murray's findings agreed with those of German and Austrian observers. D. Noel Paton, L. Findlay and collaborators were able to find no relation between the health of the mother during pregnancy and the condition of the child at birth.

For the purposes of this report deaths are classified in the following three groups: (i) Deaths due to birth asphyxia, atelectasis, birth injury or prematurity; (ii) deaths due to infective conditions; (iii) deaths due to gross developmental defects.

#### Asphyxia Neonatorum, Birth Injuries and Allied Conditions (Group I).

There were 540 deaths in Group I, that of *asphyxia neonatorum*, birth injuries and allied conditions. One

hundred and forty mature infants died after periods of a few hours to eight days after birth, though in two exceptional instances death did not occur until the fifteenth day. These 140 deaths were all due primarily to *asphyxia neonatorum*, though in several instances there was evidence of a superimposed infection such as pneumonia, bronchitis or thrombosis of the longitudinal sinus. Four hundred premature infants died primarily from asphyxia and atelectasis. In a number of these pneumonic consolidation was observed. *Post mortem* examination of the 540 infants who had died of asphyxia revealed the presence of intracranial hemorrhage in 146 instances, gross visceral hemorrhage in 62 instances and rupture of dural structures such as the *fals cerebri* or the *tentorium cerebelli* in 121 instances. Sometimes birth injuries are the actual cause of death, on other occasions they hasten death, on others they are merely incidental. It should be noted, moreover, that the asphyxial congestion existing in the new-born child enhances the effects of injury and to a certain extent also increases the liability to injury.

The pathological conditions present in all the infants who had died of asphyxia were essentially those of still-birth. The causes were antenatal and intranatal rather than postnatal, and the infants had lived only a precarious existence for the duration of a few hours or days. It is notable that nine-tenths of the deaths in this group occurred within the first week. Before the commencement of labour the tension of gases in the foetal blood and tissues is nicely balanced and the content of carbon dioxide is not so high as unduly to stimulate the respiratory centre. It is probable that during foetal life the carbon dioxide tension required to stimulate the respiratory centre is higher than at any other period, but according to Schwarz the respiratory centre becomes more irritable in the later months of gestation and the foetal blood becomes more venous owing to the gradual narrowing of the *ductus arteriosus*. Uterine contractions still further increase the carbon dioxide tension by interfering with the placental respiratory exchange. Thus the child at birth is normally in a state of partial asphyxia which gradually deepens until the carbon dioxide tension becomes sufficiently high to effect the commencement of pulmonary respiration. This theoretical explanation of the mechanism concerned in the production of respiration fits in with known facts and may be used as a working hypothesis. The probability is, however, that oxygen tension also plays an important part. Protraction of the interval between the interruption of placental respiratory exchange and the establishment of pulmonary respiration results in an increase of asphyxia and anoxæmia beyond the limits of physiological tolerance. The respiratory centre may fail when there is an excess of carbon dioxide or when there is interference with its blood supply. It is suggested that the latter may occur when pressure during birth interferes with the flow of blood to the brain.

Two other important factors in death from *asphyxia neonatorum* are engorgement of the right heart and increased intracranial pressure. In nearly all instances there is oedema of brain and meninges, and in those infants who survive for some days the oedema is very great; in these cases attacks of lividity and dyspnoea occur during life, especially during exertion.

#### Neonatal Infections (Group II).

There were 238 deaths in Group II, that of neonatal infections. Of these 120 were considered to have been due to septicæmia, but this figure must be regarded as an approximation only, owing to the difficulty of forming an opinion as to whether infection had been carried by the blood stream or not. Probably the figure errs on the low side. Pneumonia was commonly present in association with lesions elsewhere in the body. Seventy-nine of the deaths from septicæmia occurred between the end of the fourth and the end of the fourteenth day after birth.

Infection may be introduced before, during or after birth. Before the onset of labour it is rare, though, of course, syphilis is well known to be transmitted from mother to foetus *in utero* and the prenatal occurrence of various exanthemata, pneumonia and even tuberculosis

<sup>1</sup> "Child Life Investigations: The Causes of Neo-Natal Death," by J. N. Cruickshank, Medical Research Council of the Privy Council, Special Report Series, Number 145.

has been recorded. *Intra partum* infection probably occurs more frequently than was formerly believed; the rôle of the gonococcus in causing conjunctivitis is well recognized, but the extent to which other tissues, such as skin and mucous membranes, are liable to infection from the parturient canal is not clearly understood. Proof concerning the time of introduction of infection is difficult. The presence of the same organism in lesion and parturient canal does not prove that such organism gained entry during parturition. Cruickshank is inclined to agree with Hess that extrauterine infections are of much greater frequency and importance than those occurring during birth.

Most of the older authorities were of the opinion that infection was usually introduced through the umbilical stump, and even quite recently Friedländer suggested that virulent infection may enter the body through the umbilical stump without causing any noticeable local sepsis. Cruickshank found umbilical sepsis at only seven of his 800 *post mortem* examinations and he cannot agree that inflammatory lesions of the navel are the most frequent cause of neonatal sepsis, though he admits that such may have been common before the introduction of antiseptic treatment to the umbilical stump. He believes that infection is most frequently introduced by way of the skin and the respiratory tract.

Septicæmia obviously may also result from swallowing infective material from various lesions of mouth and pharynx such as aphthous and ulcerative stomatitis and injury to the buccal mucosa *et cetera*. Other portals of infection, such as ears, eyes and genito-urinary tract, are of minor importance.

#### Description of the Lesions Found.

##### Circulatory System.

There were only three instances of cardiac developmental abnormalities in the series. This is of interest in view of the frequency with which a diagnosis of congenital heart disease is used as an explanation of neonatal death. Actually such deaths are generally due to asphyxia. The heart muscle appears to be curiously immune from involvement in *sepsis neonatorum*. Cloudy swelling was observed on a number of occasions, but no actual myocarditis. The endocardium was invariably unaffected, but pericarditis as part of a septicæmic condition was observed on twelve occasions.

##### Respiratory System.

Pneumonia was the commonest infective lesion seen; it was observed on 197 occasions. The distribution and spread of the lesions in *pneumonia neonatorum* is almost invariably bronchial, but the gross appearances are often like those of lobar pneumonia in the adult. It is most common in the atelectatic lung and is especially apt to attack the lungs of feeble premature infants. One hundred and ten of the 197 infants who had died of pneumonia, had been born prematurely. The respiratory tract of the new-born infant frequently contains *liquor amnii* and meconium, and there can be little doubt that these foreign substances may predispose to pneumonia.

Lung abscess was the cause of death of nine infants in the series; in one instance death had occurred on the second day.

##### Alimentary System.

Great difficulty is attached to the diagnosis of gastrointestinal disturbances of the new-born. The term gastro-enteritis is often applied to a clinical condition in which there is no inflammatory lesion of the alimentary tract, but which is rather due to a functional disturbance of nutrition. Definite inflammation of the gastro-intestinal tract is a rarity in new-born infants. It would appear that the diagnosis of gastro-enteritis in new-born infants and in older infants and children depends on quite a different set of factors. Ulceration of the large bowel was observed on five occasions, on three of which there was a constriction of the gut below the ulceration associated with a condition of congenital megacolon.

##### Central Nervous System.

Reference has already been made to the existence of cerebral oedema and congestion in asphyxia. Though the variations in external pressure on the foetal head have been studied in relation to their effects on skull and meninges, little attention has been paid in Britain to their possible effects on the brain substance itself. Schwartz describes small hæmorrhages and areas of degeneration in the brains of infants who had died shortly after birth. He ascribes these to the effects of birth injuries. He believes the hæmorrhages to be due to low pressure on the presenting part as contrasting with high pressure within the contracting uterus; this in effect amounts to the existence of a negative pressure within the cranium, with resulting cerebral congestion and rupture of thin-walled vessels. Other observers deny the existence of hæmorrhages, save under unusual circumstances. Others, while agreeing that degenerative areas may be present, believe that these are a part of the normal histology of the brain.

Gross intracranial hæmorrhage was present in 80 mature and 81 premature infants. The age at which death had occurred varied from the first to the twentieth day and the average period of life had been less than three days. The presentations had been vertex in 113 instances, of which five had been of occipito-posterior position, face and brow two, transverse eight, breech 38. Delivery had been spontaneous in 97 instances, forceps had been applied in 31 instances, Cæsarean section had been performed once and in 32 instances some manipulative procedure had been carried out. Bleeding above the tentorium is not of such importance as bleeding below the tentorium, in which situation not only is the space more confined, but also pressure on vital centres is much more likely to occur. It is notable that in many instances of subtentorial hæmorrhage blood was prevented from passing into the spinal canal by the fact that the *foramen magnum* had become plugged by the medulla and the upper part of the cord.

Intracranial bleeding may be divided into the following types: (i) Meningeal capillary oozing, which is very common and seldom of sufficient amount to be of serious import; (ii) gross traumatic intradural bleeding caused by lesions of veins and sinuses; (iii) bleeding from the chorioid plexus into the ventricular system; this is usually traumatic; (iv) bleeding associated with disease of mother or infant; its most frequent cause is toxæmia of pregnancy and it most frequently occurs intraventricularly.

Examination of the spinal cord as a routine was not commenced until the investigation had been well advanced, so that not a great deal of evidence can be brought forward in this regard. There was no gross lesion of the vertebral column or cord in any child who had lived longer than one day. Severe injury to the cervical cord was noted on several occasions; in each instance there was a history of some difficult obstetrical operation, such as version or delivery of an impacted breech. Stoltzenberg demonstrated the connexion between injuries to the cervical cord and certain obstetric manœuvres involving lateral flexion of the head.

There were 33 instances of meningitis in the series; 29 of these occurred in association with generalized septiciæmia. Death had eventuated at periods varying from the second to the seventeenth day after birth. Some difficulty was experienced in the isolation of the offending organism. In ten instances no satisfactory bacteriological results were obtainable; in the remaining 23 coliform bacilli alone were found in 11 instances, streptococci in seven instances, pneumococci three, staphylococci one and *Bacillus pyocyaneus* one. Coliform bacilli were also present in some of the streptococcal infections. The pathogenic rôle of the coliform organisms should not be accepted without reservation.

There were 17 instances of developmental anomalies of the central nervous system in the series. In none of these was there evidence of syphilis, nor did there appear to be any relation between the anomalies and the previous health and parity of the mother.

#### Genito-Urinary System.

Hæmatoma of the *tunica vaginalis* was observed on occasions; it appeared merely to be part of the effects of birth injury. Congestion of uterus and adnexa was frequently observed and was sometimes associated with bleeding under the peritoneal coat. Congestion and minor hæmorrhages were commonly seen round the pelvis of the kidney. Gross hæmorrhages into or around the kidney were observed in eight infants, six of whom had been delivered after a difficult labour and five of whom had died on the first day. Thrombosis of renal vessels and infarction of renal parenchyma occur in association with toxæmia of pregnancy and *sepsis neonatorum*; they were observed in fifteen infants. Degenerative changes of the tubular epithelium were frequently seen, but it is doubtful whether or not these were sometimes due to *post mortem* autolysis rather than cloudy swelling. No nephritis was observed and there appears to be no indication that nephritis occurs in the new-born, save as a great rarity. When there is generalized sepsis, however, or when the infant is feeble and cachectic, the degenerative changes in the tubular epithelium may be such as to justify the term "nephrosis." Certain developmental anomalies, including hydronephrosis in eight instances, were seen. Renal calculus was observed once. It is possible that, though pyonephrosis was observed in only one instance, systematic examination of the urine of the newly-born would frequently reveal the presence of sepsis in the urinary tract.

#### Hæmorrhagic Diathesis.

The term "hæmorrhagic diathesis" is now applied only to idiopathic bleeding. The hæmorrhage is spontaneous and not preceded by any apparent symptoms. It usually commences on the second to the fourth day and ceases spontaneously in from one to three days, if it does not cause death. Rodda has concluded that the coagulation time and the bleeding time in infants are progressively prolonged from birth till the fourth day and that this is the period especially favourable to the development of the hæmorrhagic diathesis; he believes the necessary additional factor is maternal toxæmia or the administration of chloroform during labour. The evidence on these points, however, barely appears adequate for the formation of such definite opinions. There appears to be no doubt that the bleeding is due to prothrombin deficiency and is unassociated with the purpura or hæmophilia. Ten infants in the series had died after severe bleeding due to the hæmorrhagic diathesis. The mother of one of these had died shortly after delivery, of an anæmia of the pernicious type; in no other instance was there anything in the family history to account for the bleeding, nor did the mother's blood react to the Wassermann test.

#### Syphilis.

The blood of twenty-six infants in the series reacted to the Wassermann test, but *Treponema pallidum* was found in the tissues in only six instances. Similar results were obtained by Cruickshank in a previous investigation, as a consequence of which he recorded the opinion that less than 1% of infants of the hospital class in Glasgow were syphilitic. He remarked then that the Wassermann test was not dependable, as a positive reaction of the infant's blood was mostly due to transference of reacting substances from the maternal blood. The results from the present investigation are in agreement. Difficulty is frequently experienced in staining *Treponema pallidum* in tissues and inability to find the organism should not be accepted as proof of the absence of syphilis, any more than that a positive Wassermann reaction should be accepted as proof of its presence. Cruickshank points out, however, that histological and macroscopical appearances of syphilis were present in those infants in whom *Treponema pallidum* was demonstrated and absent in all others; thus his figures may be taken as approximately correct.

#### Developmental Anomalies.

Developmental defects were observed on 53 occasions; in 22 instances the defect was of such a degree as to be considered the cause of death. Abnormalities of the

central nervous system were observed on 17 occasions and included hydrocephalus which was present in 11 infants. There were 11 instances of abnormalities of the kidneys. Developmental abnormalities were sometimes merely incidental and sometimes acted as one of several causes of death.

#### Conclusions.

*Asphyxia neonatorum* and birth injuries, which account for the vast majority of neonatal deaths, are frequently due to preventable accidents incidental to delivery. This points to the necessity for stricter antenatal supervision. The public requires still more education in this regard, but the value of such education will be limited unless adequate accommodation in obstetric hospitals is available.

A plea is made for the better instruction of medical students, graduates and nurses. Formerly, in teaching students too much attention has been paid to the performance of difficult obstetrical operations and too little to the conduction of a normal labour and the supervision of pregnancy and the puerperium.

Though congenital syphilis as a direct cause of death in the neonatal period is relatively unimportant, there can be no doubt that maternal syphilis is an indirect cause of much loss of infant life from prematurity and debility. Maternal syphilis calls for greater attention from maternity hospitals and child welfare centres, where it should be treated, rather than in clinics for the treatment of venereal disease.

Efforts should be made to increase knowledge regarding the causes of prematurity which plays such an important part in neonatal death. It is suggested that ebolic drugs, failing to produce abortion, have a deleterious effect on the fetus and may well be one of the causes of prematurity.

In the prevention of infections in the neonatal period educational measures directed towards improvement in hygiene and housing are suggested. In institutions overcrowding is the condition most to be avoided.

Finally, a plea is made for continued efforts for the advance of knowledge of neonatal diseases. The application of physiological methods of investigation has met with success in *asphyxia neonatorum* and should do so in other conditions. The necessity is urged for coordination of the work of the obstetrician, the pædiatrician, the physiologist and the pathologist.

#### Treatment of Asphyxia Neonatorum.

Appended to the report is a brief discussion on modern methods of treatment of *asphyxia neonatorum*. The older methods of resuscitating the asphyxiated infant were crude and even harmful and did not depend on an appreciation of the physiological factors involved.

Carbon dioxide carried by the blood stream is the normal stimulant to the respiratory centre. In the absence of oxygen tissues cannot produce carbon dioxide. Deficiency in oxygen causes a depression of the respiratory centre which then requires more than the normal amount of carbon dioxide for the initiation of respiration. During prolonged labour decreased blood supply to the brain due to compression of the head causes a depression of the respiratory centre. The proper treatment of *asphyxia neonatorum*, then, has as its basis the administration of the normal blood gases. All violent attempts at resuscitation must be avoided. The infant should be kept warm and at absolute rest. The air passages should be cleared by suction. The lungs should then be gently inflated for two or three seconds two or three times per minute with a mixture consisting of 5% or 6% of carbon dioxide in oxygen. Inflation should be stopped immediately on the commencement of natural respiration, but inhalation of the gas mixture should be continued until full respiration is established.

When the intrathecal pressure is high, lumbar puncture is justifiable.

The feeding of enfeebled asphyxiated infants by the more usual methods is generally unsatisfactory, but it may be accomplished with a minimum of disturbance to the infant and a freedom from the risk of aspiration of food, by the employment of gavage through a small stomach tube.



While attention to the toilet of skin and mouth *et cetera* is necessary, the exposure and movement involved in bathing should be avoided until normal breathing, colour and sucking power have been attained.

If in *asphyxia livida* there appears to be danger of circulatory failure through embarrassment to the right side of the heart, bleeding from the umbilical stump is permissible. Obviously the administration of gases will be of no avail if the cardio-vascular system is incapable of conveying them to the respiratory centre. In *asphyxia pallida* shock is the predominant factor and associated with respiratory failure there is also circulatory failure. The maintenance of body temperature and the avoidance of all movement are of even greater importance in *asphyxia pallida* than in *asphyxia livida*.

## Special Articles on Diagnosis.

(Contributed by Request.)

### XXI.

#### GLAUCOMA.

SOME important diseases that are comparatively uncommon in the ordinary daily case lists of general practitioners, may on their unexpected appearance escape diagnosis on account of the possible absence of suggestive complaints from the patient or of obtrusive evidential signs. This is the more worrying to the medical attendant since he knows that delay in the institution of efficient treatment for such may result in a permanent loss or impairment of some faculty or of health. Glaucoma is of this category and may be compared to appendicitis in that it may be (i) quiet and chronic with no leading symptoms and signs, or (ii) subacute with strengthening indications of a localized disturbance, or (iii) acute with a fairly rapid or dramatically sudden onset in a patient definitely ill and suffering great localized pain.

The clinical picture at each end of the series is entirely different and the word glaucoma is, therefore, inadequate to "pelmanize" for the general practitioner the varying syndrome.

There is, however, only one underlying immediate cause for glaucoma in any form, namely, an increase in the intraocular pressure and, related to its rate of onset, degree and duration, will be the manifestations. One can deduce from this what may be expected as symptoms or signs, since the increased intraocular pressure is operating within the dense scleral globe which is unyielding, save at certain points. Compressible structures which lie approximately against it, must be compressed and the yielding parts of the wall must bulge outwards. Of these compressible structures we can for the purposes of this paper confine attention to nerves and blood vessels. The effect of pressure on the nerves will vary with their functions; thus on sensory nerves it will produce pain in different intensities, which indeed, as in all fifth nerve pain, may radiate to the other divisions of the nerve with, at the same time, possible partial anaesthesia of the cornea; on the motor nerves of the iris a slight or marked dilatation of the pupil and a slowness in contraction; on the motor nerves of the ciliary body a difficulty in accommodation, as, for example, an increase in presbyopic difficulties apparently out of proportion to the age; on the optic nerve fibres a loss of portion of the field if the peripheral fibres are involved, or a diminished visual acuteness, or an undefined intermittent difficulty on account of a paracentral scotoma if the central or paracentral fibres are involved. The fibres of the optic nerve are exposed to a double danger, since at their entrance the scleral wall tends to yield and the *lamina cribrosa* is displaced outwards. Unless it slips loosely backwards, the fibres which pass through its meshes must become attenuated and pinched and this twofold happening results in the ophthalmoscopic appearance of the "cupped disc." Pressure on the blood vessels, while helping to devitalize further the compressed nerves, may lead to the appearance of pulsating

arteries on the retina and from backward pressure (this is the most important from a general practitioner's point of view) an oedema of the cornea, while, if more sudden and intense, the general appearance of an acute congestion will be added. An oedema of the cornea is recognizable as a fine steaminess or dulness of its surface, as of a mirror lightly breathed upon. If one regards the iris-lens-iris as a diaphragm stretching across in front of the vitreous, any increase in the volume of the vitreous contents tends to displace it forward and present a shallowed anterior chamber as seen from the front. In addition to all these there is the paramount and inevitably present, less easy dimpling in of the eye ball on applying pressure to it by the fingers through the drooped upper lid.

These effects may be grouped as follows:

1. Pain in the eye and possibly also in the head.
2. Oedema and loss of sensibility of the cornea.
3. Shallowing of the anterior chamber.
4. Dilatation and sluggishness of the pupil.
5. Deterioration of vision, direct and peripheral.
6. Increased "tension" (resistance to finger pressure).
7. Cupping of the disc.

It must be remembered that these may be present only in slight or marked degree and in varying proportions.

To turn now to the more important clinical diagnosis in the consulting room, how may one expect the manifestations to be presented and what ought one to carry in one's mind as the most essential and reliable aids to that diagnosis?

#### Non-Acute Glaucoma.

In the mildest cases, that is, those with the least rise in pressure, symptoms may be almost entirely absent and the signs so slight as to be unrecognized on ordinary examination. Since patients may quite accidentally discover themselves to be blind in an eye which had given no pain and aroused no suspicion of diminishing vision and which from external appearance had not attracted the attention either of themselves or friends, and yet an increased pressure be the cause of the blindness, it follows that a patient may walk into the consulting room, since there must be some complaint to bring him there, complaining only of a little uneasiness about an eye or an uncertainty about the visual standard. There may be found no definite difference in the reading near point and the visual acuity may be about  $\frac{1}{2}$ , but if the physician is not a refractionist he does not know whether this is the natural standard for the eye or not. There may be the merest hazing of the cornea and a small difference in the size of the pupil, but if the patient has not been seen before, this difference may also be normal to the person. This is the hardest case of all to diagnose, because in others of the same type, but with a slightly higher pressure there may be more definite discomfort, a fullness, a brow ache, a headache, probably intermittent, and a visual acuity of  $\frac{1}{12}$ ,  $\frac{1}{20}$ ,  $\frac{1}{40}$  and so on. Patients may even volunteer the statement that they cannot see the nose on that side. They may say that the eye is "longer in the sight" for reading, but if both eyes should be affected, a comparison is impossible and for general purposes this sign can be regarded as of secondary importance. But no one of these signs is in itself characteristic of increased pressure only, for example, the vision and the headache may be ascribed to a possible refraction error and the pupillary change to a neurological factor. The loss of vision may even be veiled by its occurrence in an eye that never had good vision in comparison with its fellow. A shallowing of the anterior chamber, if present, is very significant, but the general practitioner will not, as a rule, commit himself beyond saying he thinks it may be shallow, but is not sure. In a little more marked case the lowered visual acuity may be found in a dull looking, insensitive eye with vessels more conspicuous than normal coursing over the sclerotic and showing a mild pericorneal flush, a pupil definitely dilated, probably ovoid vertically, sluggish and almost inactive. But if some symptoms or signs may be absent in any one case, they may just happen to be absent in the case that comes before a general practitioner, since it is not an easy case that always appears to oblige one. The question, therefore, comes to be, are there any

signs that can always be relied on to be present when there is increased pressure? One can answer in the affirmative that: (i) increased difficulty in dimpling in of the eye ball, (ii) cupping of the optic disc, (iii) alterations in the field of vision are present in all cases but in the very earliest and mildest, when, possibly, the condition could not be diagnosed unhesitatingly even by a specialist.

Only beyond these can the general practitioner be expected to make a diagnosis and if he cannot explain the symptoms he should shed himself of the responsibility by consulting an oculist who has a fuller equipment at his command. But even in the possible cases each sign has its difficulties. To know if the tension is increased, one must know, first, the average or normal resistance to the fingers and, secondly, one must apply the same method always, as is described in any text book, taking care to apply the finger above the border of the tarsal plate. With pronounced cases there is no doubt of the pressure, but to confirm the pressure in the moderate cases the two other signs may be fallen back on. Therefore, in the second place examine the optic disc. With the modern electric ophthalmoscope the cupping is more easily recognized. However, not all cuppings are pathological or glaucomatous, but the course of the vessels on the disc, as depicted in any text book, may be taken as a reliable distinguishing feature, since pallor is not to be regarded as reliable. On the whole, however, a physiological cup appears to be part of a disc of normal pinkish tint, the actual cup being whiter, whereas a glaucomatous disc is paler all over. Again, there may be a doubt as to whether a physiological cupping might be becoming pathological and it may be conjectured that, taken by itself, in such cases a diagnosis cannot rest on an ophthalmoscopic appearance alone. The third sign of defects in field may be used in corroboration and are to be searched for. For some reason it is the nasal field in one or other quadrant which shows a characteristic sign in a shaving off that tends to dip in towards the physiological blind spot. In early cases exact perimeter examination is necessary. The means for this is not supposed to be at hand for the general practitioner, but for more advanced cases an approximate test can be made with a square of white or black paper, four to five millimetres square, held on the point of a pen nib in its holder and moved on the wall or against one's coat, as the case may be, a fixation point being provided by a button *et cetera* at a distance of about one metre from the patient. In advanced cases even finger or hand movements before the fixing eye may be sufficient to elicit the field defect. In other cases the field close to the fixation point may be explored by a small test paper two millimetres square with the patient seated about one metre from the point of fixation which must, of course, be provided, as the eye must not wander in reach of the moving object. So it would appear that in all these more difficult cases, if we pass over the well advanced and therefore easily recognized ones, the diagnosis rests on the summing up by the general practitioner of a series of probabilities, putting reliance on no one isolated sign lest his restricted experience misleads him, and then deciding whether the piecing of the features together forms the picture of an eye deranged by an increased internal pressure.

#### Acute Glaucoma.

In acute cases the diagnosis may have to be arrived at differently, for the eye may be intensely inflamed, too tender for the patient to endure any prolonged examination or palpation. The less the eye is handled the better for the patient, but luckily in these cases much can be learned by rapid observation. The vision will be well below normal, the cornea steamy and the pupil, as seen through it, probably indistinctly, will be somewhat dilated and inactive or sluggish, the iris itself being muddy. The anterior chamber will also be shallow as a rule and the lids over such an eye may be congested, while the conjunctiva will be strongly injected as in an acute conjunctivitis. It is to be noted that the presence of a few brownish posterior synechiae points at the margin of the pupil does not negative glaucoma. There may be a history of some previous attack of pain for a day or two, associated with reddening of the eye and possibly haloes may have been noticed at such times around the lights at

night, but as the first attack may be the acute one presented for diagnosis, such a history may not be obtained. From the observation of these points much has been learned without distressing the patient, and the diagnosis will almost be made in the doctor's mind. All that remains is to clinch it. The defects in the field are not to be minutely searched for, so that only the estimation of the tension and appearance of the disc remain for corroboration. Through the oedematous cornea the disc may not be visible or insufficiently so for the general practitioner to grasp its details on a rapid sight and yet no more may be possible; and, indeed, if it is a primary acute attack and not one superadded on a chronic case, the disc will be hyperæmic and not "cupped" and there may be present, if it can be recognized, an arterial pulsation of the retinal arteries. The tension, therefore, may be the last remaining diagnostic hope and even this may not be easily estimated, because the lids may be considerably swollen, especially if the eye has been fomented, and the patient may shrink away at a light touch; still, the practitioner must gently try to feel down through the thickness of the lids to the globe when the increased resistance to dimpling which must be present, will be found.

It is to be remembered that acute glaucoma may be accompanied by such severe headaches with constitutional disturbances that the local origin may be overshadowed and such a diagnosis as acute influenza arrived at, and only after the general condition has improved through the administration of sedatives or from a subsidence in the severity of the pressure is the sight found to be impaired or even irretrievably lost. On the other hand, it is quite possible that an acute attack may occur in the course of a general illness and the headache be ascribed to the rise in temperature, constipation *et cetera*, and the dimness of vision to general debility. One need not think of all possible difficulties, the remembrance that such complications may arise is sufficient. Cases, however, will not always be so acute and the doctor may be able to make a reasonably good examination. Then a history of intermittent slight attack of blurring in the eye that looked red, especially around the cornea, and of haloes, may lead the thoughts in the correct direction. During such a subacute or less acute attack the eye will be red and watery, the pupil will be seen to be more oval than circular and less active, and the cornea must be finely shagreened or steamy from oedema. The eye can be palpated easily and is found to be harder than normal, and the disc, if the attack has been present for any length of time or happens to be one of a series, will certainly be cupped and the field will be affected. If the disc cannot be examined nor the field investigated, the doubt may arise as to whether an iritis or a glaucoma is before one. The injection of the conjunctiva will be the same in both cases, the iris will be discoloured in both, but the pupil in the iritis tends to be smaller than that in its unaffected neighbour and in glaucoma, on the other hand, to be larger. The shallowing of the anterior chamber is indicative of glaucoma, but an average depth does not exclude it. The cornea in glaucoma is finely roughened and in iritis, although it may look less brilliant than normal from exudate on its posterior surface, the external surface presents a more normal appearance.

I have been dealing only with cases of primary glaucoma. It is impossible to do more than mention that an increased pressure may occur as a complication of many eye injuries and even a patient returned to a country practitioner with a diagnosis of cataract or some intraocular damage, may get a glaucomatous attack and on an occurrence of pain the tension must always, as a matter of routine, be estimated. Also it must be remembered that a primary iritis may be accompanied by increased pressure and when in doubt as to diagnosis it is probably safer for the general practitioner to treat the condition as glaucoma and obtain a specialist's advice. With treatment in general this paper is not concerned, as any text book will provide sufficient information, but prognosis depends so much on early diagnosis that it has been thought worth while to deal with that only.

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# British Medical Association News.

## MEDICO-POLITICAL.

ANNUAL MEETING OF THE DELEGATES OF THE AFFILIATED LOCAL ASSOCIATIONS OF MEMBERS WITH THE COUNCIL OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

THE annual meeting of the delegates of the Local Associations affiliated with the New South Wales Branch of the British Medical Association with the Council of the Branch was held at the British Medical Association House, 137, Macquarie Street, Sydney, on October 3, 1930, Dr. E. M. HUMPHREY, the President, in the chair.

The following delegates were present: Dr. A. T. Roberts (Central Northern Medical Association), Dr. G. A. Buchanan (Central Southern Medical Association), Dr. C. W. Whiting (Central Western Medical Association), Dr. A. M. Gladden (City Medical Association), Dr. H. Hunter (Eastern Suburbs Medical Association), Dr. W. F. Simmons (Illawarra Suburbs Medical Association), Dr. L. Cowlshaw (Kuring-gai District Medical Association), Dr. R. J. Jackson (Northern District Medical Association), Dr. R. V. Graham (North-Eastern Medical Association), Dr. H. H. Lee (South-Eastern Medical Association), Dr. T. K. Potts (South Sydney Medical Association), Dr. Murray Curtis (Warringah District Medical Association), Dr. J. T. Paton (Western Medical Association), Dr. W. M. A. Fletcher (Western Suburbs Medical Association).

## Friendly Society Lodge Practice.

Two motions standing in the name of Dr. J. T. Paton in regard to the remuneration paid to medical officers of friendly society lodges were withdrawn, as it was considered that their discussion was inopportune.

## Misleading Advertisements.

DR. J. T. PATON moved:

That consideration be given to the advertisements and methods of treatment used by herbalists *et cetera* and that steps be taken to deal with the matter.

He said that as a result of the advertisements of quacks the people were relieved of large sums of money for which they received no value. He gave details of several instances. One poor person had paid one pound a week for a period of six months for treatment of talipes.

DR. W. F. SIMMONS seconded the motion and suggested that the Board of Health should take action.

DR. E. M. HUMPHREY said that the authorities would take no action and pointed out that the proprietors of newspapers accepted the advertisements.

After several delegates had quoted cases in which actual harm had been done to patients, Dr. T. W. LIPSCOMB suggested that the Country Press Association might be approached in regard to the matter of accepting advertisements from herbalists and other irregular practitioners.

The motion was carried.

## Hospital Practice.

DR. HUGH HUNTER raised the question of the system and conduct of a certain clinic in the terms of a motion standing in his name. He pointed out that according to the rules of the clinic in question members of the staff of medical officers were allowed to attend for fees the confinements of private patients whose usual medical attendant was denied the privilege of attending the patients.

During discussion it was pointed out that it would be advantageous to put the motion in general terms and it was resolved on the motion of Dr. R. V. GRAHAM:

That the system and conduct of any hospital or clinic in arranging for and allowing a member of the staff of medical officers to attend for fees the accouchements of private patients whose usual medical attendant has been refused the privilege of attending her there in spite of her expressed request, is inimical to the interests of the medical profession and should be discontinued.

## Baby Clinics.

DR. H. H. LEE moved:

That this meeting of delegates emphatically protests against the behaviour of the baby clinic nurses in visiting, unsolicited, the homes of nursing mothers and canvassing for patronage, and, further, in giving medical and surgical advice; that the Council be requested to draw the attention of the Public Health Department to these facts with a view to correcting them.

He said that the clinic authorities sent cards to the mothers. If the mothers visited the clinic, all was well. If the mother did not visit the clinic, the nurse called and asked the mother to go to the clinic. Here advice was given on the feeding of the baby; the advice was usually different from that of the attendant medical practitioner. Dr. Lee did not object to the clinic nurses canvassing for work. Dr. Lee referred in detail to certain instances in which he held that harm had been done to patients. He thought that the matter ought to be brought before the Department. He did not object to the clinics, but thought that their activities should be kept within bounds.

DR. MURRAY CURTIS seconded the motion. He said that the running of the clinics was satisfactory in the Warringah District. The members of the Warringah District Medical Association held that there should be no honoraries attached to the clinics.

DR. A. T. ROBERTS, DR. L. COWLISHAW and DR. W. F. SIMMONS supported the motion and said that the experiences in their districts were similar to those related by the delegates who had spoken before them.

DR. H. HUNTER thought that there should be no treatment of disease at the clinics. He raised the question as to whether medical practitioners had succeeded in feeding infants. Children were better fed and were healthier—the clinics had certainly done good work. He thought that the work of the clinics in looking after normal children should be encouraged.

DR. T. W. LIPSCOMB said that the cases reported by Dr. Lee would be useful to the Council of the Branch in its efforts to correct abuses. The rearing of infants was a necessary part of the activities of the State. The economic loss from unhealthy children was a big factor in the life of the country—the Government was right in looking after the health of the inhabitants. The baby clinics were part of the national health service; they were analogous to the quarantine service or any of the other services. The medical officer in charge of the clinics was an able man and a man of high ideals. He desired the cooperation of the practising members of the profession. It was not the aim of the clinics to do any treatment at all. When any departure from the normal was discovered, the nurses had instructions to send the patients to their own medical attendants. If the members would put their complaints on paper, it would be possible to bring the matter to the notice of the medical officer in charge. Dr. Lipscomb was quite sure that the officer in question would see that the regulations of his department were carried out. Dr. Lipscomb concluded by saying that he would be sorry to see the activities of the health service lessened in any way.

After DR. G. A. BUCHANAN and DR. GEORGE BELL had spoken, Dr. Lee replied. He said that while he agreed that gastro-enteritis in infants was less prevalent, all the credit could not be given to baby clinics. Improved water supply, drainage and other conditions had a great deal to do with it. He agreed with all that Dr. Lipscomb had said.

The motion was carried.



Dr. T. W. LIPSCOMB moved:

That all delegates present be asked to collect specific instances where the clinic nurses have exceeded what we believe to be their special function.

The motion was seconded by Dr. W. M. A. FLETCHER and carried.

#### Consultants.

Dr. T. K. POTTS moved:

That in view of complaints regarding unethical dealings by consultants in certain Workers' Compensation cases, all members having clear knowledge of such be asked to report them with written evidence for consideration by Council; that in cases where these irregularities are proved by Council that they be cited as infamous conduct in a professional respect before the Medical Board.

The motion was seconded by Dr. J. T. PATON *pro forma*.

After discussion by Dr. A. M. DAVIDSON, Dr. W. F. SIMMONS, Dr. J. T. PATON, Dr. T. K. POTTS and Dr. E. M. HUMPHREY it was decided with the consent of the movers to add the words, "that it be a recommendation to the Council," and it was resolved:

That in view of complaints regarding unethical dealings by consultants in certain Workers' Compensation cases, all members having clear knowledge of such be asked to report them with written evidence for consideration by Council; and that it be a recommendation to the Council that in cases where these irregularities are proven by the Council that they be cited as infamous conduct in a professional respect before the Medical Board.

#### Discussion on Insurance Cases by Members of Council concerned in a Case under Consideration.

Dr. W. F. SIMMONS moved:

That it be a recommendation to Council that any member of the Council of the New South Wales Branch who is associated with an insurance company be requested not to take part in any discussions or vote on any matter affecting the British Medical Association in its relations with insurance companies.

He said that there was nothing personal in his motion. It was felt that when a member of Council was associated with an insurance company, he was placed in a very awkward position when questions of the relationship of the Branch to the companies were under discussion.

Dr. H. H. LEE seconded the motion.

Dr. C. W. WHITING thought that it would be sufficient if the member concerned informed the Council of his relationship with the insurance company before taking part in the discussion.

Dr. W. M. A. FLETCHER concurred with the view of Dr. Whiting.

Dr. T. K. POTTS thought that the member of Council should not be deprived of the right to discussion, but that he should not be allowed to vote on the particular matter.

The motion proposed by Dr. Simmons was withdrawn and it was resolved on the motion of Dr. T. K. POTTS, seconded by Dr. W. F. SIMMONS:

That it be a recommendation that any member of the Council of the New South Wales Branch of the British Medical Association who is associated with an insurance company, be requested not to vote on any matter affecting the British Medical Association in its relation with insurance companies.

#### Votes of Thanks.

Votes of thanks were accorded to Dr. E. M. Humphrey for presiding at the meeting and to Dr. J. G. Hunter, the Medical Secretary for the Branch.

## Obituary.

### NEVILLE BAMANJI GANDEVIA.

Dr. NEVILLE BAMANJI GANDEVIA, whose death was recorded in the issue of November 8, 1930, was born on September 3, 1856. He studied medicine in England and in 1883 became a Member of the Royal College of Surgeons of England and a Licentiate of the Royal College of Physicians of London. He came to Australia in 1889 and practised in turn at Bransholme, Coleraine and Kowit; for fourteen years he practised at Surrey Hills. He was always keenly interested in military matters. He joined the Victorian Mounted Rifles in 1891 with the rank of Captain. Later, the Victorian Mounted Rifles were merged into the Light Horse. Eventually Gandevia received the Volunteer Decoration and retired with the rank of Colonel. At the outbreak of war in 1914 he volunteered for service and, holding the rank of Lieutenant-Colonel, he acted as Principal Medical Officer of various camps in Victoria and made several trips between Egypt and Australia.

His personal qualities are described by his friend Dr. Carl Dyring, who writes:

With the passing of Neville Gandevia there has been left an irreplaceable vacancy in a friendship that has existed for more than a quarter of a century. Others can tell of his success during his studentship and in his practice in spite of the obvious disability he had to carry, but when once you had the honour of being his friend, you knew it was a friendship that would only be severed as this has been. In whatever part he practised he made his patients his friends through his genial good temper and his meticulous attention to his professional duties.

I have lost a friend for whom I had the greatest affection and admiration. Perhaps the best and truest that could be said of this loyal, unobtrusive subject of the British Empire is "he was a gentleman."

GEORGE PERCIVAL STANLEY.

We regret to announce the death of Dr. George Percival Stanley, which occurred on November 24, 1930, at Kirribilli, New South Wales.

## Correspondence.

### TALIPES EQUINOVARUS CONGENITALIS.

SIR: Mr. Brockman in his monograph discounts my description as to its anatomy because I only gave details of bones from adults.

On page 103 (Walsham and Kent Hughes, 1925) there is a table of twelve talipædic tali. Two were foetal, seven from children under seven, two from children under twelve, and only one adult. The distortions in the foetus were similar in kind and degree to those in older cases. The *varus* deflection was largest in one foetus. The average of the two foetal cases was 55°, that of ten children was 45°.

The plantar deflection was largest in one foetus and smallest in the other. On a normal talus there is no plantar deflection of the neck. My method of measuring the angle was rough and imperfect, but it showed that a plantar deflection exists in every case of congenital club-foot.

The neck of every talus has a *varus* deflection. In a normal foetus it is a little less than those of the talipædic bones examined. It gradually disappears with age until it almost disappears.

This emphasizes (i) that the plantar deflection is the outstanding feature, (ii) that the *varus* deflection is largely a natural condition uncorrected. The *varus* position of the deformity, as one would expect from above, is easily cured by many methods.

Excepting all easy cases readily cured by manipulation in the first three months of life, the *equinus* is never over-

come without a wedge-shaped resection of the talus and calcaneum. I aim to obtain full dorsal flexion of the foot. Mr. Brockman has never seen a satisfactory result from a bone operation. No more have I in London. In any case that attends at regular intervals for two years, I have no hesitation in promising that I will get good dorsal flexion and a very satisfactory foot. Mr. Brockman bewails the fact that the clinics in London are full of uncured talipes. It is because the orthopaedic surgeons will not recognize the anatomical basis of the deformity. As fuller evidence of the importance of bone distortion are those recurring cases which crop up now and then, and despite careful splinting and manipulation reproduce the distortion of the neck of the talus—they require a second wedge resection.

Generally my aim is to cure the patients in about six weeks, let them walk as soon as possible, keep them under observation at regular intervals for two years. The only splint I use is a right-angled tin shoe at night and occasionally I raise the outer edge of the sole and heel of the foot. My first private case, R.J., *etatis* two (1895), jumped five feet seven inches off his talipædic foot at the Albury Sports, Christmas, 1915. That is what I call a satisfactory result.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street,  
Melbourne.  
(Undated.)

#### CLINICAL PATHOLOGISTS.

SIR: Your leader of November 15 is so much to the point that one cannot help wondering why the matter of this "injustice" has not been ventilated long ago.

However, there is another aspect of the question, which has evidently not yet come under your notice, an aspect which seems more serious, since it adds insult to injury.

There are senior consulting physicians who have seen fit to cause lay persons to be trained in laboratory work, including biochemistry. They employ such persons and also endeavour to induce other members of the profession to get their pathological work done by such lay persons, whereby they not only injure but also belittle that branch of the profession who have made clinical pathology their life's work.

There may be some excuse for sending specimens and patients for examination to the Government laboratories with the object of saving the patients extra expense, but to charge patients full pathologists' fees for work done by a salaried lay person seems unfair to the clinical pathologist and the patients alike.

Is this not a matter which might be referred to the Ethics Committee?

Yours, etc.,

"FAIRPLAY."

November 15, 1930.

SIR: I was bewildered by and amazed at your leading article entitled "Clinical Pathologists," which appeared in the issue dated November 15, 1930. So much so that I feel obliged to express my views on this subject—views which are entirely opposed to the general trend of your remarks.

I do not propose, Sir, to comment on the general tone of your article. For all practical considerations THE MEDICAL JOURNAL OF AUSTRALIA is a public newspaper. And, purely as an expression of personal opinion, I wish to say that I regard the discussion of fees payable to the members of the different branches of the profession to be a grave error in taste and distinctly *infra dignitate*. Further, I have still to learn that the function of the journal is to boost one particular section of practitioners. In contrast to the ordinarily well conceived, judicial remarks contained in the vast majority of your leading articles (if I may say so), this particular example is so different and so novel that one may justifiably conclude that it was inspired. If so, it was diabolically inspired. The object of this letter, then, is to act as an antidote to

the heretical and fallacious notions contained in that article.

At the outset let us clear our minds of cant. What, precisely, do we mean by clinical pathologists? Clinical, forsooth! It is no secret that pathologists and chemists are notoriously bad clinicians. This is no disgrace to them, but it is a fact. And I think that most clinicians will agree with me that, when the pathologists and such-like dabble in practical clinical problems, the results are always unhappy, often amusing and occasionally pathetic. Clinicians are born, not made. By acquiring correct technical habits, by assiduous practice, by faithful devotion to their tasks, such people may fully develop their inborn gifts. I believe that the same thing obtains in the practice of everyday routine pathology and biochemistry. I have trained my little dog to carry my newspaper, and he carries it very efficiently, be it today's paper, yesterday's, or last year's copy. The test tube and microscope doctors, if intelligently disposed, may learn a great lesson from my little dog. Let them keep to their laboratories, do their work in a manner that will please those that employ them, and they will reap their just reward. So let us have no more talk concerning those medical hermaphrodites, the "clinical" pathologists.

You refer to the discrepancy in salaries between the professors of pathology and bacteriology and those of medicine and surgery. This discrepancy expresses the problem in a nutshell. The part is not equal to the whole and it is surely obvious that the management of a watertight department like bacteriology is a very different proposition to the supervision of a department whose activities cover the whole realm of the principles and practice of medicine.

In actual fact there is no need for the private pathologist. The system is wasteful, inefficient and altogether unsatisfactory. Pathologists should be employed by big institutions at a high salary, and the fees from paying patients should go towards the upkeep of their departments.

But, as things are at present, the private pathologist must come back to earth and, if he is wise, he will take to heart what the clinicians—the vast majority of total practitioners—really think about him and his position in the universe. These views I summarize as follows:

1. In recent years there has been a tremendous and quite unjustifiable exaltation of function of the pathologist and biochemist. He is simply an aid to diagnosis and a check on the results of treatment and on the progress of the condition of the patient.
2. In the actual handling of patients, especially private patients, pathologists, as a class, are usually *gauche*, sometimes frankly uncouth, and generally upsetting to the sick. Nursing sisters, the world over, will bear ample testimony to this.
3. For the services rendered pathological fees are too high. If this state of things continues, there is no reason why half a dozen practitioners in the suburbs or in the cities and towns of the Commonwealth should not combine and employ a technician to carry out the essential work.
4. A great deal of the pathologist's work is pure routine, and by no stretch of the imagination can be called scientific. Yet many of these laboratory doctors, openly or occultly, according to temperament, assume that they, and they alone, have the scientific outlook on clinical medicine. This is not the case. No doubt they can jump through a few chemical hoops in a more skilful manner than the ordinary medical man. But with all their tricks they are not more "scientific" than the keen physician, surgeon, or general practitioner, though the scientific outlook of the latter group may be masked by the compulsion to cultivate the much despised though highly desirable "bedside manner."
5. Pathologists belong to one of the sheltered group of practitioners. That is to say, they have no reputation to make with the public. They miss most of the hurly-burly and competition associated with the practice of medicine. As a direct result of this they miss much of the worry, responsibility and heartaches that go with practice.

Yours, etc.,

"DR. JEKYLL."

November 20, 1930.

### THE FUNCTION OF THE GALL BLADDER.

SIR: When putting forward some views on gall bladder disease in your journal of October 4 the various functions assigned to the gall bladder, more particularly that occurring in paragraph 1, page 465, were those one so often hears quoted by clinicians.

It is my opinion that the healthy gall bladder has a very definite function as a reservoir for bile, but only in a concentrated form.

I wish to thank Professor Osborne for his criticism in the journal of the eighth instant and for his valuable reference.

Yours, etc.,

H. BULLOCK.

155, Macquarie Street,  
Sydney.

November 18, 1930.

### Books Received.

SICK CHILDREN: DIAGNOSIS AND TREATMENT. A MANUAL FOR STUDENTS AND PRACTITIONERS, by Donald Paterson, B.A., M.D., F.R.C.P.: 1930. London: Cassell and Company Limited; Sydney: Angus and Robertson. Crown 8vo., pp. 538, with sixteen half-tone plates and eighty-five figures in the text.

### Diary for the Month.

- Dec. 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.  
Dec. 2.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.  
Dec. 2.—New South Wales Branch, B.M.A.: Hospitals Committee.  
Dec. 3.—Victorian Branch, B.M.A.: Annual General Meeting.  
Dec. 4.—South Australian Branch, B.M.A.: Council.  
Dec. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.  
Dec. 11.—New South Wales Branch, B.M.A.: Branch.  
Dec. 11.—Victorian Branch, B.M.A.: Council.  
Dec. 12.—Queensland Branch, B.M.A.: Branch (Annual).  
Dec. 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.  
Dec. 19.—Queensland Branch, B.M.A.: Council.

### Medical Appointments.

Sir Colin MacKenzie (B.M.A.), Dr. M. J. Holmes (B.M.A.), Dr. L. W. Nott (B.M.A.) and Dr. A. G. Butler (B.M.A.) have been appointed as members of the Medical Board for the Territory for the Seat of Government under the provisions of the *Medical Practitioners' Registration Ordinance, 1930*.

Dr. V. H. Webster has been appointed Acting Resident Magistrate for the East Kimberley Magisterial District and Acting Magistrate of the Local Court, Wyndham; also Acting Chairman of the Wyndham-East Kimberley Court of Session, Western Australia.

Dr. E. J. T. Thompson (B.M.A.) has been appointed Acting Inspector-General of the Insane, Western Australia.

### Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xvi.

HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Junior Resident Medical Officer.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Medical Superintendent.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Vacancies.

### Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Hospital. Mount Isa Mines.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

### Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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